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The American Heart Journal

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Original Communications

THE THERAPEUTIC VALUE OF QUINIDINE IN CORONARY THROMBOSIS COMPLICATED BY VENTRICULAR TACHYCARDIA*

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THE treatment of coronary thrombosis is often mainly concerned with proper care of the complications that occur after the onset of the attack. In fact when the course of the illness runs smoothly, as it often does, apart from the complete rest given to the patient no other therapy is necessary. One of these complications which is not at all infrequent is of such a nature that the difference between accurate recognition of the situation with the institution of proper therapy and failure to do this might easily make the difference between life and death of the patient. This complication is the inception of paroxysmal ventricular tachycardia.

Paroxysmal ventricular tachycardia is distinctly less common than paroxysmal auricular tachycardia, but unlike the latter is generally an indication of grave heart disease. Scott,[1] however, reported an exceptional instance of this disorder in a patient who was otherwise well, and he also emphasized the value of quinidine in preventing the attacks of ventricular tachycardia from returning. Jones and White[2] and Gilchrist[3] reported similar cases, and Levine and Curtiss[4] used quinidine for ventricular tachycardia in a case of mitral stenosis having advanced myocardial failure. In general, the most common condition in which ventricular tachycardia occurs is coronary occlusion. This has been noted both clinically[5] and experimentally.[6, 7]

When this complication develops it can present a difficult problem in therapy, inasmuch as it will not respond to the usual procedures that are employed. From clinical experience it has been found that digitalis will not control the attacks of ventricular tachycardia, and there is some reason to believe that it may even tend to perpetuate the

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arrhythmia. In fact, digitalis can produce this arrhythmia when given in sufficient doses.[8, 9] Although when ventricular tachycardia occurs in patients suffering from coronary thrombosis it may be transient and disappear after a few hours, the following case illustrates that it can be persistent. Under these circumstances it becomes a complication of such great importance that the life of the patient is endangered because of this fact alone, entirely apart from the other features that are involved in so serious an underlying disease. The case reported below, it seems, would have ended fatally if the extremely rapid heart rate had not been controlled, and the success obtained from the use of quinidine sulphate warrants its further trial under similar circumstances.*

REPORT OF CASE

The patient was a carpenter of fifty-three years who entered the hospital March 24, 1927, complaining of severe precordial pain. The family and marital history were not important. Twenty years ago he had a severe attack of articular rheumatism. Eight years ago he had an attack of empyema. For the past year he was slightly short of breath on going up hill or when hurrying, and for the past six months there was a chronic cough. There was no edema of the ankles and no pain in the chest, although there had been a moderate degree of substernal tightness associated with the dyspnea.

Present Illness.—He was admitted to the hospital by ambulance at 1:25 A.M. on March 24. At nine o'clock that evening, four and one-half hours before admission, while sitting quietly at home, he was taken suddenly with a severe pain in the precordium and a sense of compression across the sternum with the pain radiating down both arms. He became very weak, collapsed, and was put to bed, but did not lose consciousness. He was seen by one of us (S. A. L.) in consultation three hours after the attack. At this time he was still complaining of pain, had just vomited, and had a blood pressure of 158/100; the heart sounds were very distant and there was a distinct gallop rhythm. He received forty-five milligrams of morphine subcutaneously in fifteen milligram doses before entering the hospital.

Physical Examination.—On entrance he was found to be a muscular man propped up in bed, still complaining of pain about the sternum. He looked seriously ill, had a greyish pallor, had difficulty in breathing, and felt sure he was going to die. The respirations were forced and shallow (rate 30). The skin was clammy with a cold sweat, especially about the head. The pupils did not react because of the morphine. The thorax was somewhat enlarged in the anteroposterior diameter; expansion was poor but equal on both sides. The apex impulse of the heart was neither seen nor felt, but the heart was slightly enlarged to percussion. The sounds were regular, rate 110, of tick-tack quality with a distinct gallop rhythm. (Fig. 1.) There were no murmurs or thrills. The pulses were thready in character, the arterial wall was palpable but not calcified. The blood pressure was 130/80. The lungs showed a few cracking râles at both bases, and there were no signs of free fluid. The abdomen was slightly distended and tympanitic, the liver edge was not felt and there was no tenderness or dullness in the flanks. There was no peripheral edema; reflexes were normal. Temperature by rectum was 100.5° F., by mouth, 99.7°. White cells, 12,800 per c.mm.; hemoglobin (Tallqvist), 95 per

*Quinidine sulphate has been used in three other cases of coronary thrombosis showing ventricular tachycardia, and in each instance it produced a normal sinus rhythm.

cent; red cells, 5,072,000 per c.mm.; differential count normal; urine essentially negative. The picture was typical of coronary thrombosis.

Course in the Hospital.—For the first week he ran the usual course of a severe coronary thrombosis. He had almost continual pain that had to be controlled with morphine; he complained of difficulty in breathing and fear that he was going to die. The râles at the bases of the lungs increased so that they extended above the angle of the scapula on both sides. The liver was never palpable, nor was there any tenderness in this region. There was no peripheral edema. Rectal temperature remained elevated from 100° to 102° F.; pulse dropped to 100, with respirations of

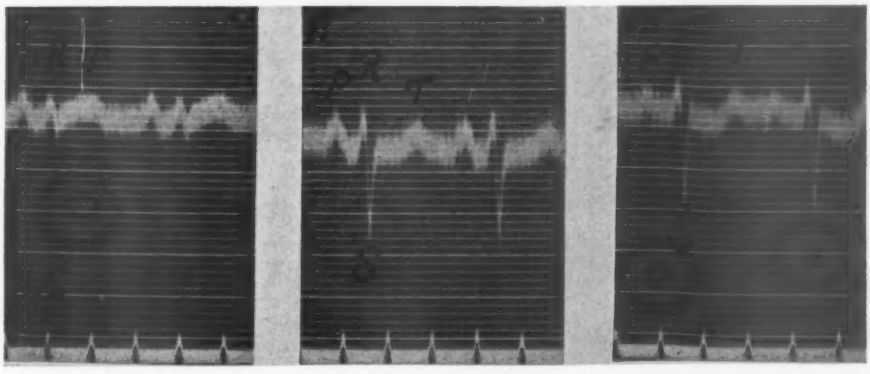

Fig. 1.—March 31, 1927. The customary three leads show minor changes in the R-T interval suggestive of a recent cornary thrombosis. Note the rounding of the R-T interval in Lead I.

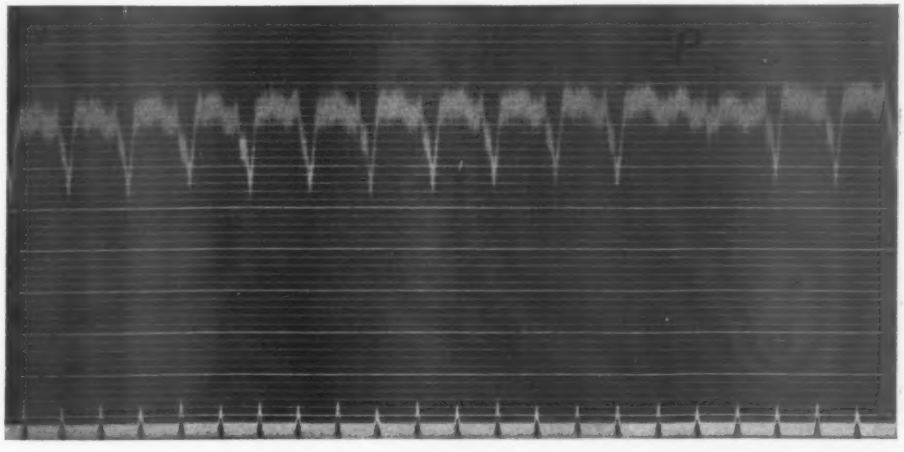

Fig. 2.—April 9, 1927. Lead I. Before any quinidine was given. Rate about 200. Condition is one of rapid ventricular tachycardia with a rare interruption of a normal supraventricular beat. (P-wave.) Note the difference between the normal and abnormal ventricular complexes.

25. He ran a persistently high white blood cell count of from 11,000 to 14,000. The blood pressure dropped further, ranging from 96/74 to 110/82. During the second week there was some clinical improvement. He required less morphine for the pain and restlessness, seemed to breathe more easily, and there was no increase in râles at the bases. His temperature did not go above 100° by rectum, but reached that level every day; pulse remained 100 and regular; respirations, 20 to 25. Blood pressure showed a slight rise, again varying between 110/82 and 122/84 on daily observations. The white count dropped to between 8,700 and 10,700.

On April 7 (the fourteenth day in the hospital) the patient had an attack of transient auricular fibrillation during which there was considerable prostration, but in about an hour he had completely recovered, and the heart had returned to a normal rhythm.

On the next morning, April 8 (the fifteenth day in the hospital), during the examination of the heart an unusual condition was found. The heart sounds were much louder than they had been at any time during our observation, and at first it was thought that there was a rate of 100 with a marked gallop rhythm. On more prolonged examination it was made out that the rate was really 198 at the apex, with sounds that, although loud, were fetal in character. The rhythm was essentially regular, but on careful auscultation slight irregularities could be detected, and occasionally the intensity of the first sound changed. No murmurs were heard. The pulse was very poor in quality, absolutely irregular, slow (rate 80), and the beats varied markedly in intensity. No blood pressure reading could be obtained. Although the patient still seemed critically ill, aside from the heart findings no striking change in his condition as compared to the past few days could be noted. There was no increase in cyanosis or dyspnea, and no further evidence of congestion. He did seem somewhat weaker and more apprehensive, but he was not conscious

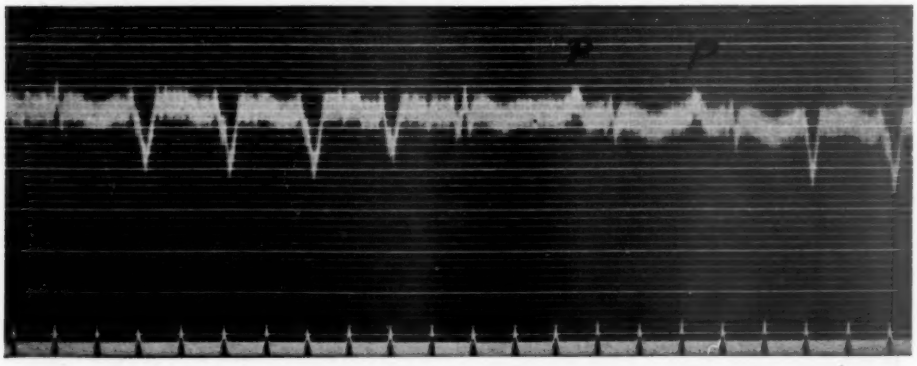

Fig. 3.—April 12, 1927. Lead I. After 1.2 grams of quinidine sulphate had been given. Note the appearance of more frequent normal supraventricular beats and the partial intermission of the ventricular tachycardia. The rate of the ventricular tachycardia itself has been slowed from 200 (Fig. 1) to 150 per min.

of palpitation. It was suspected that the patient had developed ventricular tachycardia, and this was confirmed by an electrocardiogram which showed a heart rate of 200 (Fig. 2).

For reasons stated below it was decided to give quinidine sulphate and in view of the seriousness of the situation the first dose was given intravenously a little over twenty-four hours after the onset of the attack. Electrocardiograms which were taken frequently during the injection of 0.3 gram of quinidine sulphate* showed no change at all in the rhythm or form of the complexes.

Two hours after this procedure the picture was further complicated by the occurrence of a sudden excruciating pain in the left lower leg and foot, with the blanching of the foot from the ankle down. No dorsalis pedis pulsation could be felt on this side, although it was present on the right, and the foot became numb and cold. There had obviously been an arterial occlusion by a thrombus or embolus. Heat in the form of a cradle with electric lights giving a temperature of 85° F. was placed over the leg within an hour and kept on constantly for several weeks.

Following the failure of the intravenous quinidine to affect the rhythm, it was

*One and two-tenths grams of quindine sulphate were dissolved in 28 c.c. of distilled water to which 4 drops of dilute sulphuric acid (U.S.P.) were added and the solution autoclaved for twenty minutes at fifteen pounds pressure.

not known exactly what dosage would be required to break the extreme rapidity of the heart, and the condition was too urgent to permit trial doses with slow increases thereof from day to day. At 4:30 P.M. on April 9 he was given 0.4 gram of quinidine sulphate by mouth but no effect was noted. At noon the next day 0.5 gram was given. Throughout these days frequent electrocardiograms were taken to follow the mechanism of the heart rhythm. Doses were rapidly increased at intervals of several hours giving 0.6, 0.7, 0.8, 1.0 gram until the afternoon of April 12, when, after a dose of 1.2 grams, a distinct effect was produced (Fig. 3). It was now noted that the quinidine was interrupting the ventricular tachycardia

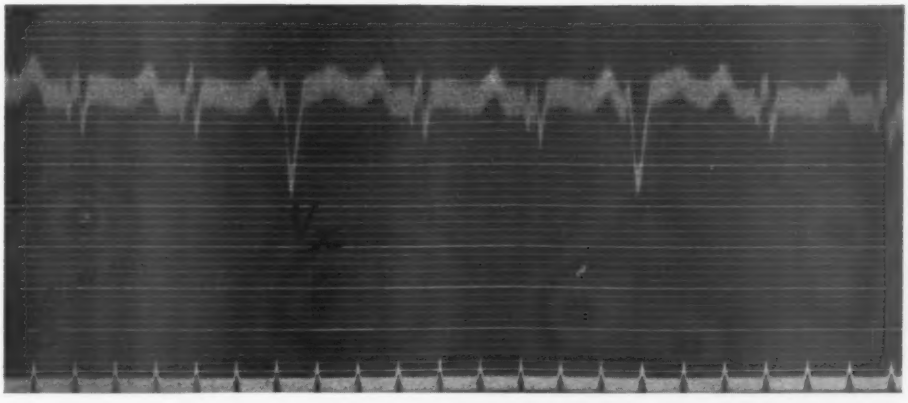

Fig. 4.—April 12, 1927. Lead I. After 1.5 grams of quinidine sulphate. Now there is only an occasional interruption of the normal mechanism by a ventricular extrasystole (Vx).

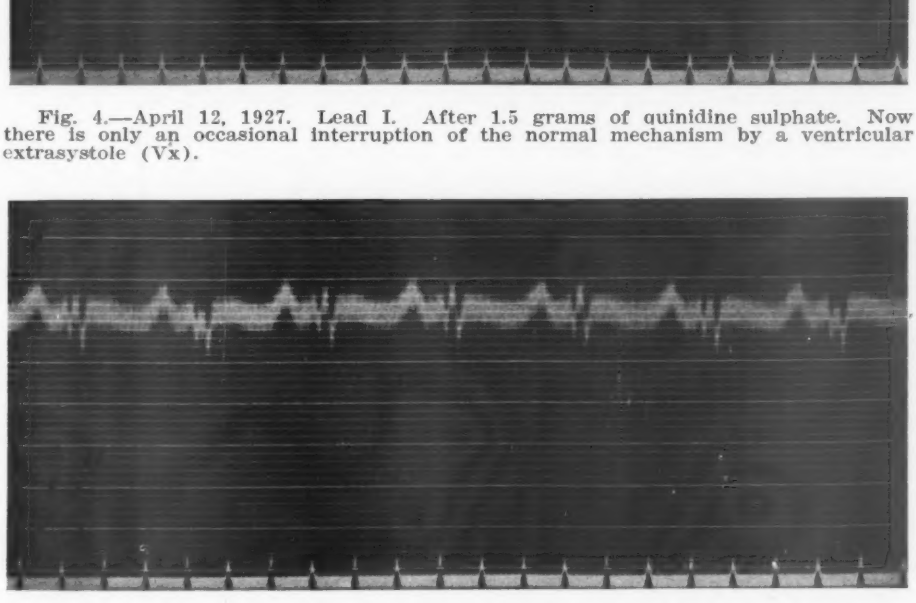

Fig. 5.—April 15, 1927. Lead I. While 1 gram of quinidine sulphate was given from 4 to 5 times a day. Note complete restoration of normal rhythm.

and allowing frequent interpolations of normal supraventricular beats. In addition the rate of the abnormal ventricular beats had slowed from 200 (Fig. 2) to 150. This indicated a specific effect of quinidine on the ventricular mechanism. It was then decided to give doses of 1.5 grams every several hours, and in the evening of April 12, after two doses of 1.5 grams of quinidine several hours apart, the heart had become slow, and there were only occasional ventricular extrasystoles. (Fig. 4.)

At this point the question came up as to whether to continue such large doses of quinidine frequently or to omit the drug. It was noticed that the extrasystoles tended to reappear, and the patient was therefore given 1.0 gram of

quinidine sulphate four or five times a day until April 18. In this way the heart rhythm was made regular (Fig. 5), and it remained normal even after the drug was omitted entirely on April 18. At this time he had had a normal rhythm for five days and there was no further return of any irregularity.

During the four days when the heart was very rapid the clinical condition of the patient seemed always desperate. The pulse was barely felt; no blood pressure reading could be obtained; cyanosis became increasingly more marked, and the heart sounds were almost inaudible. When the change to the slow rate occurred on the afternoon of the 12th of April, the heart sounds distinctly improved in quality, and for the first time in four days there was no pulse deficit and the radial pulse was of fair quality.

Coincident with the return to a normal rhythm there was a marked general improvement in his clinical condition. In addition, the left foot which had been blue and cold and painful and apparently going on to gangrene began to show evidence of increased warmth and return of normal color. The patient remained in the hospital until May 19, making a complete stay of eight weeks. There was a gradual improvement, and on discharge the only complaint was moderate pain in the left foot. Five months after the occurrence of his coronary thrombosis he is alive, able to be up and about the house somewhat, and complaining only of slight pain in the left foot when he tries to walk on it.

DISCUSSION

The rationale of the use of quinidine in ventricular tachycadria has an experimental basis in the work of Drury, Horsfall, and Munly[10] who applied the same methods of measuring the variations of the refractory period of the ventricular muscle of the dog under quinidine as had previously been used by Lewis et al[11] in measuring the effect of the drug on the auricular muscle. They found a distinct lengthening of the refractory period in the ventricular muscle when quinidine was administered, showing that in this respect quinidine affects the auricular and ventricular muscle of the dog in essentially the same way. Scott[1] has shown that digitalis, atropine, nitroglycerin, and adrenalin have no action in ending an attack of ventricular tachycardia.

The clinical problem that was presented in the case reported was very unusual. There prevails a general timidity concerning the use of quinidine in patients suffering from serious heart disease and this hesitation we feel is well founded because of the occasional serious result following the use of the drug.[12] In most instances there is an alternative course that the physician might follow with considerable hope of success, namely, digitalis. This is the situation when the condition to treat is auricular fibrillation. In this case the situation was quite different. Ventricular tachycardia had developed in a case of severe coronary thrombosis and persisted. We knew that it could not be controlled by digitalis and the gravity of the condition was so great that the use of the only medication that might restore the heart to normal rhythm was indicated despite the risk involved. Quinidine sulphate was therefore administered, and it is important to appreci-

ate that unusually large doses were necessary to produce the desired effect. It was only after 1.5 grams were given five times a day that the normal rhythm was maintained. The only other group of cases where such large doses were given was reported by Sidel and Dorwart,[13] and here the indication was to control auricular fibrillation. It is also significant that considerable aid was obtained from taking frequent electrocardiograms, for when the dose of 1.2 grams of quinidine sulphate had been reached, and we wondered whether we should continue increasing it, the heart tracing showed that a partial effect had been already obtained, and this gave us courage to administer such doses as we had never given before.

SUMMARY

A case is reported of coronary thrombosis which developed a persistent ventricular tachycardia with a heart rate of 200. This lasted for several days, and the clinical condition seemed desperate. Quinidine sulphate in extremely large doses proved successful in restoring the heart to a normal rhythm. We believe that this beneficial effect was a specific result of the medication and that it probably was life saving.

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ANATOMICAL STUDIES ON THE CORONARY ARTERIES AND THEIR BRANCHES*

I. Arteria Anastomotica Auricularis Magna

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THE blood supply to a tissue is often a key to the understanding of pathological processes which may occur in the tissue. The vessel to be described supplies blood to several important structures in the heart which are often the seat of pathological processes, and it is hoped that its description in detail may furnish a clue to the pathogenesis of some of the lesions found in the mitral and aortic valves, and possibly, in the base of the aorta.

Kugel and Gross[1] called attention to a conspicuous blood vessel which produces a free anastomosis between the left and right coronary arteries, runs in the interauricular septum for part of its way, and furnishes blood vessels at times to the aortic cusp of the mitral valve. This paper is concerned with a more detailed description of the course, structure, and function of this vessel together with its possible rôle in the pathogenesis of valvulitis and lesions of the commissures and of the base of the aorta.

MATERIAL AND TECHNIC

Fifty normal human hearts have been studied in detail and to this have been added observations on a number of hearts which were the seat of valvulitis.

The accompanying list gives the number of hearts of each decade studied.

1st	decade	3
2nd	"	5
3rd	"	3
4th	"	6
5th	"	10
6th	"	13
7th	"	6
8th	"	3
9th	"	1
		50

These hearts were injected with barium sulphate gelatin under standardized conditions by the Gross method.[2] They were subsequently rendered transparent and dissected. Microscopic sections were taken where indicated.

*From the Laboratories of the Mount Sinai Hospital, New York City.
†George Blumenthal, Jr. Fellow.

ANATOMY OF THE ARTERIA ANASTOMOTICA AURICULARIS MAGNA

As will be seen from the description and the statistical observations which follow, this vessel was found invariably in the fifty specimens studied. Like the main coronary arteries and their branches, it is subject to variations, and, as will be seen, these variations fall into three types. Because of the large calibre of this vessel, the important rôle which it probably plays in the pathogenesis of valvulitis, and because of its constant occurrence and site, it will be referred to as the *arteria anastomotica auricularis magna.*

The following review of the anatomy of the interauricular septum and its relationship to the surrounding structures is given in order that the somewhat complicated pathway of the arteria anastomotica auricularis magna may be more readily understood and also because in this way the probable function of this vessel may be more easily understood.

The anterior portion of the root of the aorta is inserted into the left ventricle and the interventricular septum and abuts against the posterior surface of the root of the pulmonary artery (Fig. 1). The right lateral portion of the root of the aorta is inserted into the interventricular septum. In its posterior and left lateral aspects the root of the aorta becomes inserted into the base of the aortic cusp of the mitral valve. In this region, too, it is intimately associated with the insertion of the tricuspid valve at the angle formed between the septal and anterior cusps of the latter.

Furthermore, it is to be remembered that whereas the posterior portion of the interauricular septum sits on top of the interventricular septum, it divides anteriorly forming a "Y," in which the stem of the "Y" is represented by the interauricular portion posteriorly. The two wings of the "Y" form the anterior walls of both auricles, and enclose the roots of the pulmonary artery and the aorta. The right wing of the "Y" has inserted into it the anterior cusp of the tricuspid valve. The left wing of the "Y" has inserted into it the aortic cusp of the mitral valve. *It is this left wing of the "Y" together with the stem which, in the great majority of cases, carries the arteria anastomotica auricularis magna.*

The variations of this vessel can be placed in three general groups:

1. Where it forms a simple anastomosis between the left circumflex coronary artery or its branches and the *posterior portion* of the right circumflex coronary artery or its branches.

2. Where it forms a simple anastomosis between the left circumflex coronary artery or its branches and the *anterior portion* of the right circumflex coronary artery or its branches.

3. Where in the greater part of its course this vessel is represented by diffuse anastomoses between branches from the anterior portions

of the left and right circumflex coronary arteries and the posterior portion of the left circumflex coronary artery.

In the *first* and most common variation (33 out of 50 hearts in this series) the arteria anastomotica auricularis magna arises from the left circumflex coronary artery (or its branches) one to two centimeters below its mouth and plunges directly posteriorly into the wall of the left auricle (Figs. 1 and 2). This stout artery pursues a wavy course mesially within the anterior wall of the left auricle, one-half to one centimeter above the insertion of the aortic leaflet of the mitral valve. It then proceeds backward in the interauricular septum (stem of the "Y") for its entire length, and near the crux* of the heart unites

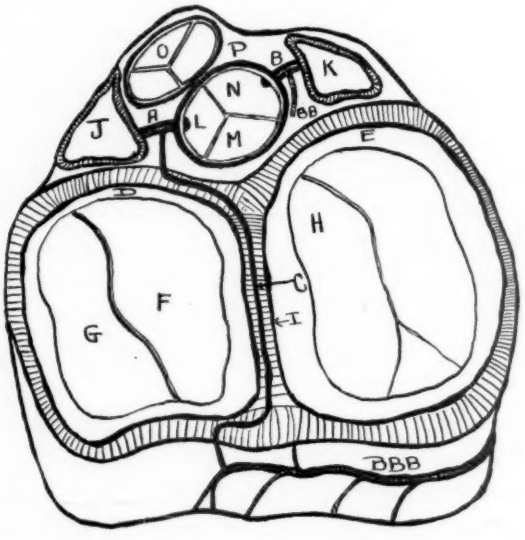

Fig. 1.—Diagram of the cross-section of the heart in the region of the auriculo-ventricular ring, showing the course of the arteria anastomotica auricularis magna (first variation).

Key to Fig. 1: *A*, left circumflex coronary artery (anterior portion); *B*, right circumflex coronary artery (anterior portion); *BB*, branch to the sino-auricular node (ramus ostii cavae superiores); *BBB*, posterior distribution of the right circumflex coronary artery; *C*, arteria anastomotica auricularis magna; *D*, anterior wall of left auricle; *E*, anterior wall of right auricle; *F*, aortic leaflet of the mitral valve; *G*, posterior cusp of the mitral valve; *H*, septal leaflet of tricuspid valve; *I*, interauricular septum (stem of "Y"); *J*, left auricular appendage; *K*, right auricular appendage; *O*, pulmonary artery; *P*, aorta. (*L*, left posterior cusp; *M*, right posterior cusp; *N*, anterior cusp.†) *R*, arcuate vessel (aborted form); *S*, crux of the heart.

directly or through the intermediary of smaller twigs with the posterior distribution of the right circumflex coronary artery or its branches (such as the ramus septi fibrosi, posterior descending coronary artery, etc.). Small twigs from the auricular branches of both coronary arteries may join this vessel. In those instances where the left circumflex coronary artery rounds the margo obtusus and passes beyond the

*By "crux" of the heart is meant that portion of the heart where the two auricles and the two ventricles meet posteriorly.

†This is the terminology used in Gray's "Anatomy."

crux onto the right heart, the arteria anastomotica auricularis magna reenters the left coronary artery posteriorly.

In 7 cases in our series the artery to the sino-auricular node (Ramus ostii cavae superioris) arose from the left circumflex coronary artery, and in these cases the arteria anastomotica auricularis magna was given off from this branch or formed a part of it.

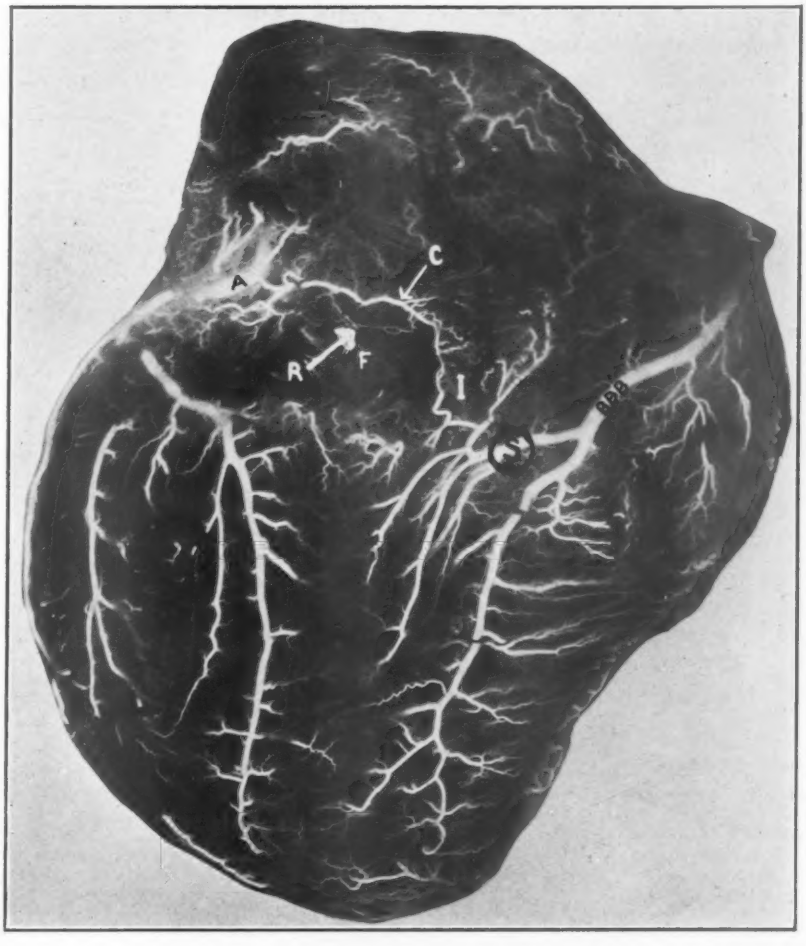

Fig. 2.—Photograph of injected and cleared heart (posterior view), showing the course of the arteria anastomotica auricularis magna (first variation). The posterior wall of the left auricle has been cut away. (See Fig. 1 for key to lettering.)

In the *second* variation (13 hearts in this series) the arteria anastomotica auricularis magna forms an anastomosis between the anterior portion of the left circumflex coronary artery and the *anterior* portion of the right circumflex coronary artery (Figs. 3 and 4). Here again the arteria anastomotica auricularis magna arises from the anterior portion of the left circumflex coronary artery or its branches, and plunges directly inward and backward within the anterior wall of the left auricle to reach a point within the interauricular septum (stem

of the "Y") about two to three centimeters anterior to the crux of the heart. Here it abruptly turns on itself anteriorly, retraces its course through the stem of the "Y," and passes within the anterior wall of the right auricle (right wing of the "Y") to join the anterior portion of the right circumflex coronary artery or its branches.

In the *third* variation (4 cases in this series) the arteria anastomotica auricularis magna arises in the anterior portion of the left coronary artery as in the first two variations; very soon, however, it breaks up into a number of branches (Fig. 5). As these approach the stem of the "Y," they are met by anastomotic branches coming from the anterior portion of the right coronary artery through the anterior wall of the right auricle (right limb of the "Y") and by branches coming anteriorly through the interauricular septum from the crux of the

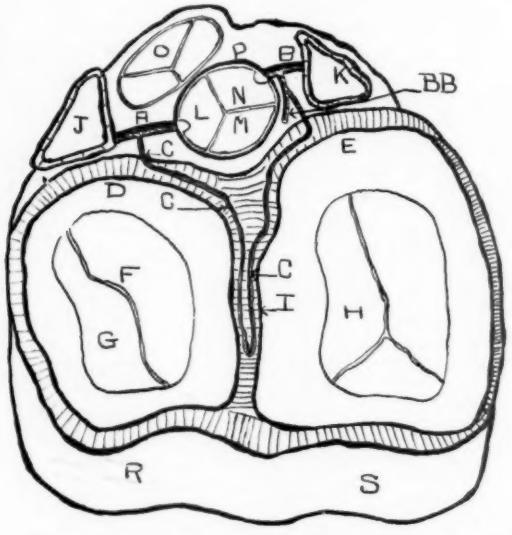

Fig. 3.—Diagram of the cross-section of the heart in the region of the auriculo-ventricular ring, showing the course of the arteria anastomotica auricularis magna (second variation).

Key to Fig. 3: *A*, left circumflex coronary artery (anterior portion); *B*, right circumflex coronary artery (anterior portion); *BB*, branch to the sino-auricular node (ramus ostii cavae superiores); *C*, arteria anastomotica auricularis magna; *D*, anterior wall of left auricle; *E*, anterior wall of right auricle; *F*, aortic leaflet of the mitral valve; *G*, posterior cusp of the mitral valve; *H*, septal leaflet of tricuspid valve; *I*, interauricular septum (stem of "Y"); *J*, left auricular appendage; *K*, right auricular appendage; *O*, pulmonary artery; *P*, aorta. (*L*, left posterior cusp; *M*, right posterior cusp; *N*, anterior cusp.) *R*, right ventricle; *S*, right ventricle; *T*, arcuate vessel (aborted form).

heart. The latter branches may arise from the terminations of the left or right coronary artery, depending upon which of these passes the crux of the heart posteriorly. It may be considered that, inasmuch as in this variation the anastomoses are rather diffuse, it is unjustifiable to speak of an arteria anastomotica auricularis magna. Generally, however, a larger trunk can be seen among these anastomotic branches. Moreover, even in this type it is preferable to retain the name for descriptive purposes.

RELATION OF THE ARTERIA ANASTOMOTICA AURICULARIS MAGNA TO THE BLOOD SUPPLY OF THE AORTIC LEAFLET OF THE MITRAL VALVE

As was described in a previous publication,[1] when blood vessels exist in the aortic leaflet of the mitral valve, they almost invariably arise from the arteria anastomotica auricularis magna. When this

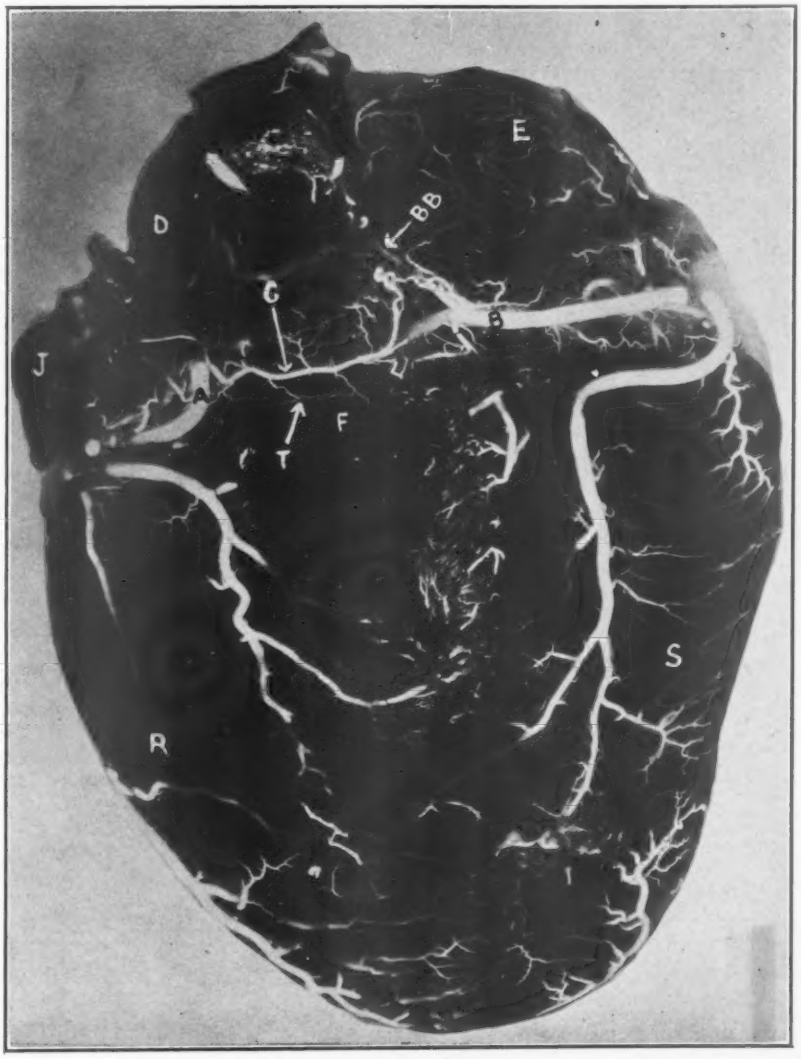

Fig. 4.—Photograph of injected and cleared heart (posterior view), showing the course of the arteria anastomotica auricularis magna (second variation). The posterior walls of the left and right auricles and part of the left ventricle have been removed. (See Fig. 3 for key to lettering.)

valve leaflet is not vascularized, branches are seen to descend from the arteria anastomotica auricularis magna toward the base of the aortic cusp of the mitral valve, where they terminate (Figs. 2 and 4). Here they may be joined by an arcuate vessel. These vessels appear to

be in all respects analogous to the arteriae valvulares* which at times supply the aortic leaflet of the mitral valve. Indeed the architectural form of these vessels so closely simulates the arteriae valvulares of this valve flap that the impression is gained that one is dealing with arteriae valvulares in an aborted form.

This impression is further strengthened by the fact that these vessels, together sometimes with their arcuate arch, have been seen in several specimens to penetrate the valve leaflet and terminate within

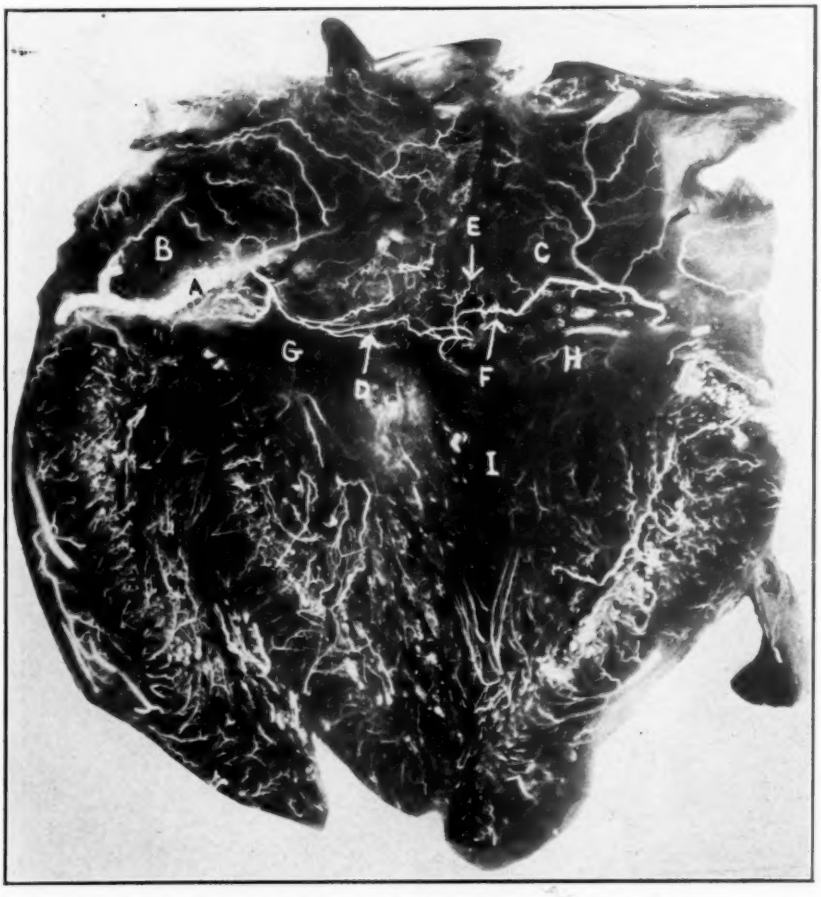

Fig. 5.—Photograph of injected and cleared heart, showing the anterior view of the left ventricle and left auricle, revealing the diffuse distribution of the arteria anastomotica auricularis magna (third variation).

Key to Fig. 5: A, left circumflex coronary artery; *B*, anterior wall of the left auricle; *C*, interauricular septum; *D*, arteria anastomotica auricularis magna; *E*, anastomotic branches going to the anterior wall of the right auricle (right wing of the "Y"); *F*, branches from the interauricular septum (stem of the "Y"); *G*, aortic cusp of the mitral valve; *H*, posterior cusp of the mitral valve; *I*, interventricular septum; *J*, anterior wall of the left ventricle.

its substance, at times close to the base of the valve leaflet, at times deeper down. It would seem, therefore, that all gradations exist between this arch in the aborted form and in the form which has been described as mitral valve vasculature of the "complete" type.

*Blood vessels of the valves. See Kugel and Gross[1].

RELATION OF THE ARTERIA ANASTOMOTICA AURICULARIS MAGNA TO THE BLOOD SUPPLY OF THE CUSPS OF THE AORTIC VALVE, OF THE COMMISSURES AND OF THE AORTA

As the arteria anastomotica auricularis magna passes through the anterior and medial walls of the left auricle, it bears an intimate relation to the left and the right posterior cusps of the aortic valves (Figs. 6 and 7). When the blood vessels occur in the right and left posterior

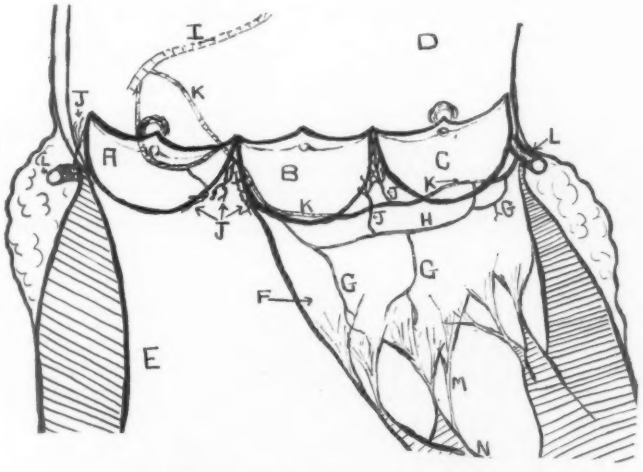

Fig. 6.—Diagram of aortic cusps and ventricular surface of the aortic cusp of the mitral valve, showing the relationship of the arteria anastomotica auricularis magna to the blood supply to the aortic cusp of the mitral valve and to the commissures.

Key to Fig. 6: *A*, anterior cusp of the aortic valve; *B*, right posterior cusp of the aortic valve; *C*, left posterior cusp of the aortic valve; *D*, aorta; *E*, left ventricle; *F*, ventricular aspect of aortic leaflet of mitral valve; *G*, arteriae valvulares to mitral valve; *H*, arcuate vessel (aborted form); *I*, branch to the sino-auricular node (ramus ostii cavae superiores); *J*, blood vessels to the commissures; *K*, arteria anastomotica auricularis magna; *L*, left circumflex coronary artery; *M*, chorda tendineae; *N*, papillary muscle.

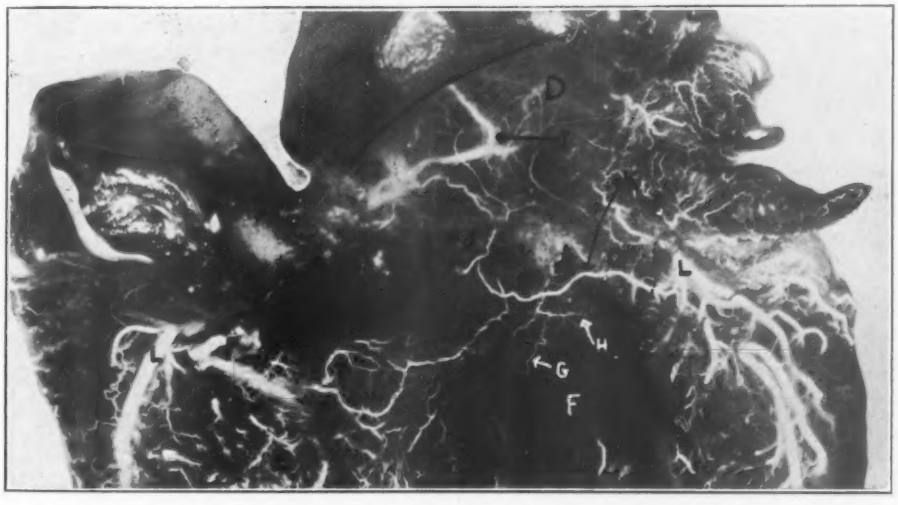

Fig. 7.—Photograph of injected and cleared specimen, showing the relationship of the arteria anastomotica auricularis magna to the aortic cusps. Fig. 6 is a diagrammatic representation of this photograph. (For lettering see Fig. 6.)

cusps of the aortic valve, they arise from this anastomotic vessel and enter the cusps, usually at the base near the center (Fig. 8). The valvular vessel then proceeds upward to the corpus arantii where it dichotomizes and sends fine branches along the line of closure. The blood supply to the right anterior cusp of the aortic valve is usually

Fig. 8.—Diagram of the right and left posterior cusps of the aortic valve, showing the relationship of the arteria anastomotica auricularis magna to the cusps of the valve and the origin of the blood supply to these and to the commissures.

Key to Fig. 8: *A*, arteria anastomotica auricularis magna; *B*, aborted vessels to base of aortic leaflet of the mitral valve; *C*, vessels to cusps of aortic valve; *D*, corpus arantium; *E*, line of closure; *F*, vessels to the commissures and to the base of valve; *G*, right posterior cusp of the aortic valve; *H*, left posterior cusp of the aortic valve.

Fig. 9.—Photograph of the right and left posterior cusps of the aortic valve, showing the arteria anastomotica auricularis magna and the origin of the blood supply to the commissures and to the aortic leaflet of the mitral valve.

Key to Fig. 9: *A*, right posterior cusp of the mitral valve; *B*, left posterior cusp of the mitral valve; *C*, arteria anastomotica auricularis magna; *D*, aborted arch; *E*, arteria valvulares; *F*, ventricular view of the aortic leaflet of the mitral valve; *G*, commissure and its vascular supply.

derived from the myocardial branches of the left circumflex coronary artery in this area, but it may also come from the arteria anastomotica auricularis magna.

The anastomotic artery also sends a few branches forward, upward, and inward which dichotomize and terminate in fine capillaries between the cusps of the aortic valve (commissure) chiefly between the left and right posterior cusps, and to a lesser extent between the right posterior and anterior cusps (Figs. 8 and 9). These fine twigs, particularly in their capillary distribution, send small branches to the insertion of the valve cusps along the insertion line. Furthermore, the auricular branches in the region of the posterior surface of the aorta may send fine twigs downward and anterior to terminate in the commissures.

The anastomotic vessel in its course is also in intimate relationship with the posterior and left lateral surfaces of the aorta. To this area it sends many vessels which anastomose diffusely with the auricular branches of the right and left coronary arteries and with the anterior branches to the aorta which arise from the right and left coronary arteries.

There is then an intimate relationship between the blood supply of the mitral valve and that of the commissures and the aortic valve leaflets, as well as with the posterior portion of the root of the aorta.

DISCUSSION

From the description which has been given of the arteria anastomotica auricularis magna, it is easily seen that we are dealing with a vessel which furnishes a wide anastomotic channel between the left and right coronary arteries and, if for no other reason than this, deserves a detailed description. Though it may be a coincidence, it is of interest to note that in several hearts which were the seat of arteriosclerotic disease the arteria anastomotica auricularis magna was of unusually large calibre. This goes hand in hand with the observations made by Gross[2] to the effect that under similar conditions other anastomotic channels, particularly in the interventricular septum, appear to be unusually wide.

The fact that blood vessels of a fairly characteristic form and course descend from this vessel toward the base of the aortic cusp of the mitral valve and that all gradations have been found between this picture and one where the vessels pierce this valve flap and terminate within its substance at various distances toward the closure line, serves as additional evidence to favor the view that the arteriae valvulares are not of inflammatory origin. It is also seen that the arteria anastomotica auricularis magna gives rise to blood vessels which (when they exist) supply the cusps of the aortic valve, and also the aortic commissures.

The latter fact is exceedingly important, inasmuch as we may have here an explanation of at least one of the factors concerned with the

mechanism of the localization of lesions in the commissures, such as those found in rheumatic disease, subacute bacterial endocarditis (Lewis and Grant[3]), and syphilis.

SUMMARY

A large anastomotic blood vessel which runs in the auricular walls and links up the left and right coronary arteries has been described. This vessel is constant in its occurrence, though subject to variations in course. It supplies branches to the aortic cusp of the mitral valve, to the aortic valve (when blood vessels are found in these sites), to the commissures, and to the base of the aorta.

Its intimate relationship with these structures suggests that it may play a part in the pathogenesis of mitral and aortic lesions, commissural lesions, and possibly lesions at the root of the aorta.

I wish to thank Dr. Louis Gross for the aid and inspiration he has given me in this work.

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AN EPIDEMIC OF RHEUMATISM AT A CARDIAC CAMP

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IN SUBMITTING the following survey of the incidence of rheumatism and its allied diseases at a camp for convalescent cardiac patients, we wish to emphasize that rheumatism may assume the proportions of an epidemic in its manifestations. The cases of rheumatism herein considered were patients with frank joint pains associated with fever, elevation of the pulse rate, and limitation of motion of the joints involved. The joint symptoms were frequently preceded by abdominal pain, tachycardia, or slight elevation of temperature; rheumatic nodules were not observed in any of the cases.

Sunset Camp is situated sixty miles from the city of Chicago on a small inland lake and is comprised of three residential frame buildings with four dormitories and accommodations for forty-five ambulatory heart patients. The dormitories are steam-heated, and the camp is provided with adequate supplies for the comfort and care of the children. The staff consists of a resident physician, three nurses, a visiting physician, and an advisory board of cardiologists* from Chicago. The camp has been in existence for five years, with uniformly good results in over 60 per cent of the cases. The children are recruited from the various cardiac dispensaries of Chicago and are kept at the camp for periods varying from six to eight weeks, provided no intercurrent illness requires their return to the city. The age limits are from eight to fifteen years. The girls' session has been conducted during the months of May and June and the boys' session during the months of September and October.

The spring season of 1927 presented many variations from other camp seasons in that the greatest number of complications arose during this session. Thirty-eight girls arrived at the camp on April 29, 1927; five more came a few days later. Bad weather was encountered from the outset; it was unusually cold and rainy. Within the first five days, twelve cases of upper respiratory infection developed; however, this number did not exceed the usual incidence at the opening of previous camp seasons.

On May 5, 1927, the first case of acute rheumatic infection made its appearance. Nine more cases of rheumatism, one of chorea, and one of acute tonsillitis developed during the course of the following four weeks. The incidence and progression are indicated in Table I.

*During the course of this epidemic, Doctors Harrold A. Bachmann and Walter W. Hamburger served as consultants to the camp.

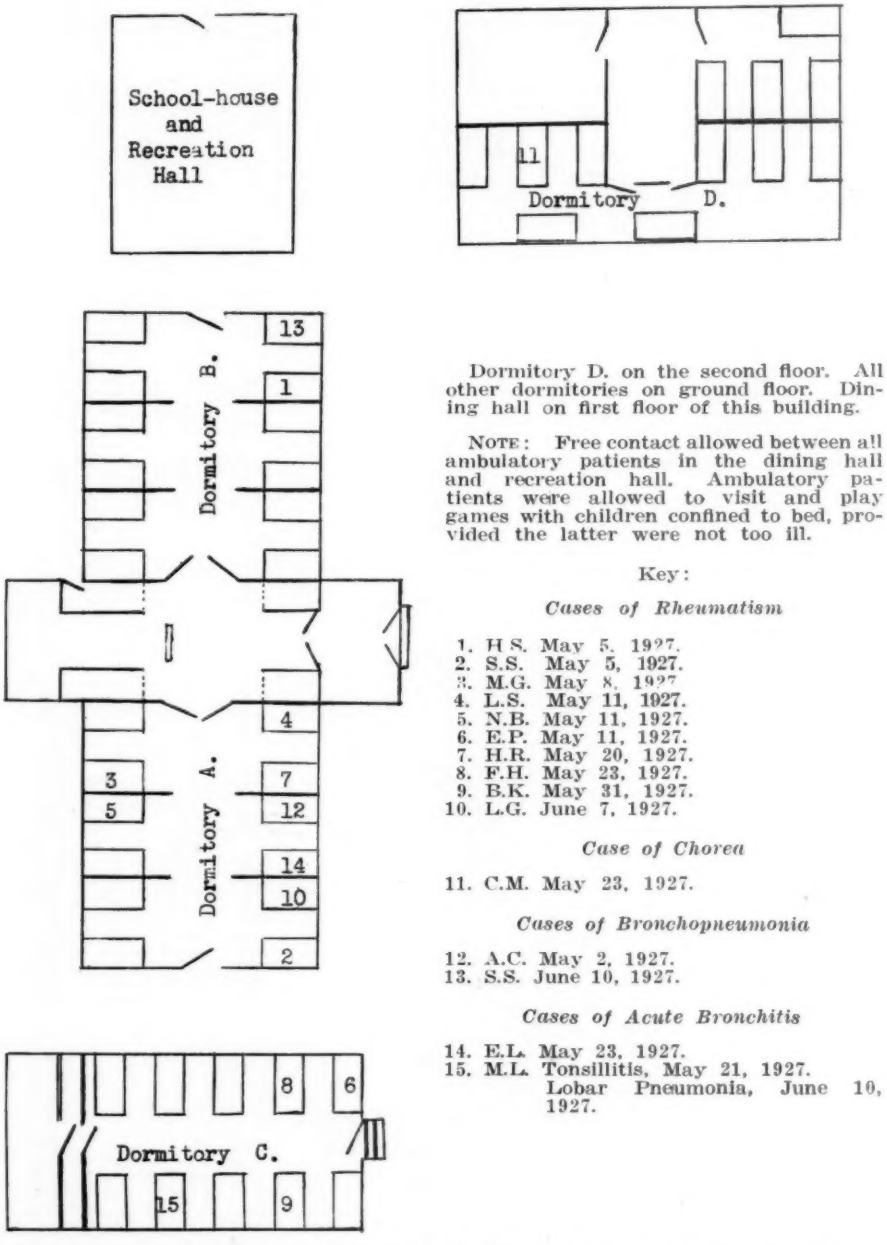
TABLE I

DATE OF ONSET	PATIENT	CLASSIFICATION	AGE	EXISTENT CARDIAC LESION	ETIOLOGY OF THAT LESION	TONSILS	TEETH	PROGRESS AND REMARKS
May 5	H.S.	I	13	Mitral disease	Rheumatism 2/21/27 3/ 7/27	Out; tags present	O.K.	5/ 5/27 Rheum. in left shoulder; rhinitis 5/ 9/27 Left wrist 5/11/27 Signs of fresh heart involvement 5/13/27 Right elbow and shoulder 5/21/27 Returned to Chicago 6/17/27 Died; Cook County Hospital
May 5	S.S.	IIA	11	Mitral disease	Rheum. first at age of 5½. Pains in shoulders, knees and hands frequently during past year, especially when weather changed	Tags on both sides	O.K.	5/ 5/27 Rheum. in knees; rhinitis. Intermittently in both knees. 5/17/27 Rheum. still present 5/21/27 Returned to city
May 8	M.G.	IIB	12½	Mitral disease	Tonsillitis October, 1916. Many previous attacks of tonsillitis. Chorea. 1923	Out June, 1917 Tag of Left tonsil	O.K.	(Note: Influenza during three weeks prior to admission) 5/ 8/27 Rheumatic pains in both shoulders; right trapezius region 5/10/27 Active heart involvement 5/13/27 Salicylates stopped, pains return 5/15/27 Pain in neck 5/17/27 Pain in shoulders. No joint pains thereafter 5/25/27 Returned to city; active carditis
May 11	N.B.	IIB	13	Congenital heart-block	Congenital	Out 1925 Tags	O.K.	5/11/27 Right knee 5/12/27 Both knees 5/13/27 Precordial pain, no change in heart 5/16/27 Occasional precordial pain 5/23/27 Pain in chest. Temperature 103.6° F. 5/25/27 St. Luke's Hospital

TABLE I—CONT'D

DATE OF ONSET	PATIENT	CLASSIFICATION	AGE	EXISTENT CARDIAC LESION	ETIOLOGY OF THAT LESION	TONSILS	TEETH	PROGRESS AND REMARKS
May 11	L.S.	IIA	13	Mitral disease	Tonsillitis before removal in 1919. Rheum. at ages of 5, 7, and 11	Tags	O.K.	5/11/27 Pain in left arm (muscle) 5/12/27 Left wrist and knee 5/13/27 Left knee, pain and swelling 5/14/27 Both knees 6/ 9/27 Shifting pains in joints continue; abdominal pain No further joint pain 6/15/27 Home. Signs of mitral stenosis present
May 11	E.P.	IIA	13	Mitral disease	Rheum., 6 or 7 attacks since 1922	Out	O.K.	5/11/27 Left wrist 5/13/27 Left wrist and arm 5/15/27 Right shoulder No pains after 5/16/27
May 20	H.R.	IIA	11	Mitral disease; extra systoles	In hosp. 5 times with heart disease. Rheum. etiology.	Tags	O.K.	5/20/27 Left knee, one day only. Temp. 99.2° F.; Pulse 108
May 23	F.H.	IIA	14	Congenital	Congenital	Out	O.K.	5/23/27 Right shoulder 5/24/27 Abdom. L.U.Q. Temp. 99.4.° Pulse 108 5/28/27 Right knee 6/ 4/27 and 6/5/27 Right scapular region. No temp. or pulse variations
May 30	E.K.	IIA	10	Mitral disease	Rheum., 1922 twice. 1923 once	Left tag	Caries	5/30/27 to 6/2/27 Right arm near elbow
June 7	L.G.	IIB	12	Double mitral and aortic disease	Tonsillitis at 2. Scarlet fever, 1923 Rheum., 1923	Left tags	Caries	6/ 7/27 Right arm 6/15/27 Pain in right arm persistent; sent home
May 26	C.M.	IIA	7	Mitral disease	Chorea, 1925 and 1926	Tags	Caries	5/21/27 (Chronic otitis media); chorea noted. Slight improvement at end of camp season
May 21	M.L.	I	12	Mitral disease and pericarditis	Rheum., 1923, pericarditis, 1924-1925, 5 months	Present	Caries	5/21/27 Acute follicular tonsillitis until 5/25/27. Kept in bed. 6/10/27 Bilateral lobar pneumonia Died 6/21/27

The etiological factors contributing toward the existing heart lesions in the ten cases that developed rheumatism at camp were as follows: rheumatism, 7; chorea and tonsillitis, 1; congenital heart disease, 1;



Dormitory D. on the second floor. All other dormitories on ground floor. Dining hall on first floor of this building.

NOTE: Free contact allowed between all ambulatory patients in the dining hall and recreation hall. Ambulatory patients were allowed to visit and play games with children confined to bed, provided the latter were not too ill.

Key:

Cases of Rheumatism

1. H.S. May 5, 1927.
2. S.S. May 5, 1927.
3. M.G. May 8, 1927.
4. L.S. May 11, 1927.
5. N.B. May 11, 1927.
6. E.P. May 11, 1927.
7. H.R. May 20, 1927.
8. F.H. May 23, 1927.
9. B.K. May 31, 1927.
10. L.G. June 7, 1927.

Case of Chorea

11. C.M. May 23, 1927.

Cases of Bronchopneumonia

12. A.C. May 2, 1927.
13. S.S. June 10, 1927.

Cases of Acute Bronchitis

14. E.L. May 23, 1927.
15. M.L. Tonsillitis, May 21, 1927. Lobar Pneumonia, June 10, 1927.

Fig. 1.—Schematic representation of the incidence and relationship of cases.

congenital heart disease and tonsillitis, 1. The etiology for the existing heart lesion in the patient that developed acute follicular tonsillitis was rheumatism; and that for the patient that developed chorea was chorea.

Among the entire group of forty-three, the etiology of the cardiac lesions was as follows: rheumatism, 35; chorea, 3; scarlet fever and chorea, 1; congenital heart disease, 1; congenital heart disease and tonsillitis, 1; no etiology elicited, 2.

The cardiac lesions in the group that developed rheumatism were as follows: mitral disease, 7; mitral and aortic disease, 1; congenital heart-block, 1; congenital heart disease (patent ductus arteriosus), 1. The patient that developed chorea had a mitral lesion, and the patient with tonsillitis had mitral and pericardial involvement.

Carious teeth were present in four of the twelve patients who became acutely ill, two of these four being in the rheumatic group.

The patient with acute follicular tonsillitis was the only one that had not had a tonsillectomy. Of the remaining eleven, seven had tonsillar regrowth present. In two cases the fossae were clean.

All twelve patients were ambulatory and well compensated at the outset. They were classified as follows: rheumatic group: IIA, 6; IIB, 3; I, 1; acute tonsillitis case, I, chorea case, IIA.

The physical relationship of cases according to beds in the dormitories is indicated in the accompanying diagram. (Fig. 1.)

On May 5, 1927, one case of rheumatism appeared in each of the dormitories designated *A* and *B*. Subsequently five new cases appeared in Dormitory *A* at varying intervals, although three days seemed to be the common interval. No new cases appeared in Dormitory *B*. On May 11, 1927, when two cases appeared in Dormitory *A*, the first case in Dormitory *C* occurred. Subsequently two cases of rheumatism and one of acute follicular tonsillitis developed at irregular intervals in Dormitory *C*. The case of chorea first appeared on May 21, 1927, in Dormitory *D*.

The course and follow-up of the cases discloses the following facts: One patient (H.S.) died June 17, 1927; the patient with acute follicular tonsillitis died June 20, 1927, of bilateral lobar pneumonia. The other patients have all shown varying degrees of cardiac involvement, but at the present time are free from active infection.

While no accurate diary was kept of the weather and temperature conditions, the accompanying summary taken from the U. S. Weather Reports from a station eighteen miles from camp indicates that during the month of May, 1927, there were a greater number of rainy days and a smaller number of clear days than for the same month during the preceding four years. The average temperature, however, was a little higher than for previous years. The month of June, when compared with that of the preceding four years, shows a smaller number of rainy days and thermometric conditions which compare favorably with these years. It is interesting to note in this connection that nine out of our ten cases of rheumatism, the case of tonsillitis, and the case of chorea all developed during the month of May, 1927.

TABLE II

YEAR	NO. OF CLEAR DAYS	NO. OF CLOUDY OR PARTLY CLOUDY DAYS	NO. OF DAYS WITH PRECIPITATION OF 1/100 INCH OR MORE	DAILY TEMPERATURE			GREATEST DAILY RANGE OF TEMPERATURE	PERCENTAGE POSSIBLE SUNSHINE. U. S. WEATHER STATION AT CHICAGO (60 MILES FROM CAMP)	
				HIGHEST	LOWEST	MEAN			
1923	16	15	4	78	30	52	37	68%	For the month of May
1924	12	19	12	82	28	51	43	62%	
1925	18	13	7	90	29	52.2	44	70%	
1926	14	17	11	87	27	56	47	72%	
1927	6	25	14	87	35	55	42	45%	
								Normal 64%	
1923	14	16	9	97	45	68.6	31	73%	For the month of June
1924	6	24	18	88	36	60.6	33	68%	
1925	11	19	10	94	40	68.6	37	73%	
1926	12	18	11	86	37	61.5	32	75%	
1927	12	18	7	96	41	62.4	35	70%	
								Normal 71%	

From U. S. Weather Station eighteen miles from camp.

DISCUSSION

For many years attempts have been made to isolate a specific virus as the causative agent of rheumatic fever. Although the organisms isolated have not as yet been recognized as specific, the feeling is universally prevalent that rheumatic fever is an infectious disease. The organisms described by Poynton and Paine,[1] and more recently by Small[2] and by Birkhaug[3] have been streptococcal in character. Most observers suspect the nasopharynx as the portal of entry.

Atwater[4] in his studies on the epidemiology of the disease has concluded that it is one of the communicable infections. Boas and Schwartz[5] in a review of the incidence of rheumatism among 53 children with rheumatic heart disease stressed the transmissibility of the rheumatic virus. They surveyed the literature and concluded from their experience and that of others "that rheumatic fever has many of the characteristics of other infectious diseases with a low degree of contagiousness but that at times, and under special circumstances it may assume epidemic proportions."

In their series, however, a "bout of rheumatic fever was ushered in by a bronchopneumonia" in a number of the cases. In our group, two cases of pneumonia occurred as indicated on the diagram, but neither was followed by rheumatic involvement. The patient with tonsillitis, who later died of bilateral lobar pneumonia, had a pericardial rub during her pneumonia, but no evidence of rheumatism was noted. Since no post-mortem examination was permitted, the nature of the pericardial involvement could not be determined.

Andrieu[6] described rural epidemics in and about Aucun in 1925-1926 and an outbreak in the garrison troops at Toulouse in 1924-1925. His findings were "that relatively long and intimate contact between the sick and the well is necessary for the transmission of the infection; the contagion is only by interhuman contact; that the incubation period is still undetermined; that abortive forms are common; that there are convalescent and recurrent carriers of the virus; that immunity is not developed by an attack of rheumatic fever. He also stressed presumed contributing factors of poverty, overcrowding of living premises, including the effects of dampness, cold and fatigue."

He also recommended the institution of personal and family prophylaxis, isolation of cases, the disinfection of oral discharges, and the disinfection of the rhinopharynx.

Since acute rheumatic fever is not a reportable disease in Chicago, we could gain no accurate information regarding its incidence in the city during the months that the camp was in session. Although facilities for accurate bacteriological study were not available at our camp, we were able to make accurate clinical observations. Daily temperature and pulse records were kept for all of the children, and in all bed

cases similar observations were made four times a day. Regular physical examinations, leucocyte counts, and urinalyses were also made. From a survey of our camp records for previous seasons, both spring and fall, we find that in spite of the occurrence of upper respiratory infections in abundance and occasional cases of arthralgia without fever, tachycardia, or further heart involvement, there was only an isolated case of rheumatic fever. This season, however, yielded a group of cases of rheumatic fever which varied from the mild abortive type to the more severe forms; all cases showed varying degrees of further cardiac damage. The group as a whole differed in no way from groups of previous seasons as to methods of selection, all cases of active infection being refused. Twenty of the children had been at previous camp sessions.

Although Miller[7] recently, and most writers in the past, have emphasized the importance of cold and dampness, and although the appearance of our cases coincided fairly well with the inclement weather, we feel that the regular progression of our cases and the fairly restricted incidence as to dormitories points to the occurrence of an epidemic in which communicability was a factor.

SUMMARY

1. Ten cases of rheumatism, one of chorea, and one of acute tonsillitis occurred within a short period in a group of children at a cardiac camp.

2. Unusually inclement weather coincided fairly well with the incidence of the cases.

3. Although the weather undoubtedly played a part in their incidence, we feel that communicability was also a factor.

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EXPERIMENTAL MYOCARDITIS*

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EXPERIMENTAL lesions of the myocardium have been produced and described by a number of observers. Fleisher and Loeb[1] found that a single, nonlethal injection of caffeine and adrenalin sufficed for the production of myocarditis in the rabbit, and they analyzed in detail the effect of such injections. We employed the same method as these investigators. In all, 47 rabbits were given each a single intravenous injection of 0.025 gm. of caffeine sodium benzoate in a 1 c.c. watery solution, followed several minutes later by an injection of 0.2 c.c. of a 1 to 1000 solution of adrenalin (Parke-Davis). These injections were administered slowly, as previous observers have shown that fewer animals die of acute edema of the lungs when the injections are so given. While our work in general confirms the observations of Fleisher and Loeb, we have extended it in various directions and, in particular, we have included in our statistical data a lesion in the papillary muscle of the left ventricle which these authors described but did not consider in their statistics. Furthermore, we observed a lesion in the left auricle which had been overlooked by previous investigators.

MYOCARDIAL CHANGES

In order to rule out the possibility that these may have been spontaneous lesions and in order to analyze the functional effects of the myocardial changes, a number of rabbits were examined electrographically before and after these injections. The results have been published separately.[2] We may state here that these myocardial changes are all accompanied by changes in the electrocardiographic records, none of which were observed in rabbits not previously injected with caffeine and adrenalin solutions.

Location of the Lesion.—Forty-seven rabbits were injected with the caffeine and adrenalin solution. They were then killed at various periods ranging from a few minutes to twenty weeks following the injections. Of these animals, forty showed either a gross or microscopic lesion in the heart. The positions of the lesions and the relative number of rabbits affected were as follows:

1. In the papillary muscles of the left ventricle, 97.9 per cent.
2. In the anterior aspect of the upper half of the left ventricle, 64 per cent.

*From the Department of Pathology, Washington University School of Medicine, St. Louis, Mo.

3. In left auricle, 45 per cent.
4. In posterior aspect of upper half of left ventricle and left auricle, 29 per cent.
5. In posterior aspect and in upper quadrant of left ventricle, 9.5 per cent.
6. In upper quadrant of anterior aspect of left ventricle, 9.5 per cent.
7. In middle third of anterior aspect of left ventricle, 6 per cent.

In no case was the right side of the heart involved. This fact has also been observed by previous investigators. Furthermore, the lesion never affected the entire left ventricle. The apex of the left ventricle showed a pathological change only in one case, and this was considered to represent a preexisting lesion. The average lesion appears as a pale, yellow-brown plaque which shades off rather abruptly into the reddish-brown color of the surrounding normal myocardium. The plaque is slightly raised and firm and is, on the average, 6 to 8 mm. wide and about 1 cm. long. Its upper end usually encroaches upon the auriculoventricular junction and extends slightly into the left ventricle. The upper half of the papillary muscles is pale and firm, and the base of the papillary muscles is usually encroached upon by the lesion in the left ventricle. There is, however, considerable variation from this average lesion.

Animals that were examined at autopsy before the heart had stopped beating showed in some cases that part of the myocardium which was involved in the lesion in the left ventricle, was definitely noncontractile; furthermore, the left ventricle contracted as a whole at a slower rate than is the case in normal animals. When the left auricle was affected, the contractions were sluggish, and the auricle apparently was unable to empty itself.

Character of the Myocardial Lesions: A. *The Development of the Lesion.*—Examination of the heart immediately following the death of the animal from acute edema of the lungs, occurring as a result of the caffeine and adrenalin injections, reveals the left auricle and left ventricle firmly contracted, while the right auricle and ventricle are widely dilated. The myocardium of the left ventricle and auricle are considerably paler than normal. The muscle of the right heart is normal in appearance although the chambers are dilated.

The typical gross myocardial lesions have been seen as early as forty-eight hours following the injection of caffeine and adrenalin, although they are not marked at this time. The majority of the rabbits show the gross lesion in six days, but the maximum gross change becomes visible in from twelve to twenty-one days following injection. Eight to ten weeks following injection about 25 per cent of the rabbits showed the gross lesion, while after twenty weeks only 17 per

cent showed it. The disappearance of the gross lesion can be explained on the basis of the microscopic studies of hearts at the various intervals of time.

B. *The Microscopic Changes.*—In animals that die *within ten minutes* following injections of adrenalin and caffeine, the microscopic changes are not pronounced. Interstitial edema is usually found, but not to a marked degree. The cross striations of the muscle fibers are not as distinct as in the noninjected animals, while the muscle fibers of the left ventricle are wider than normal. This condition is not due to a true hypertrophy but may be the result of the contracted state of the myocardium, or, to edema of the muscle fibers.

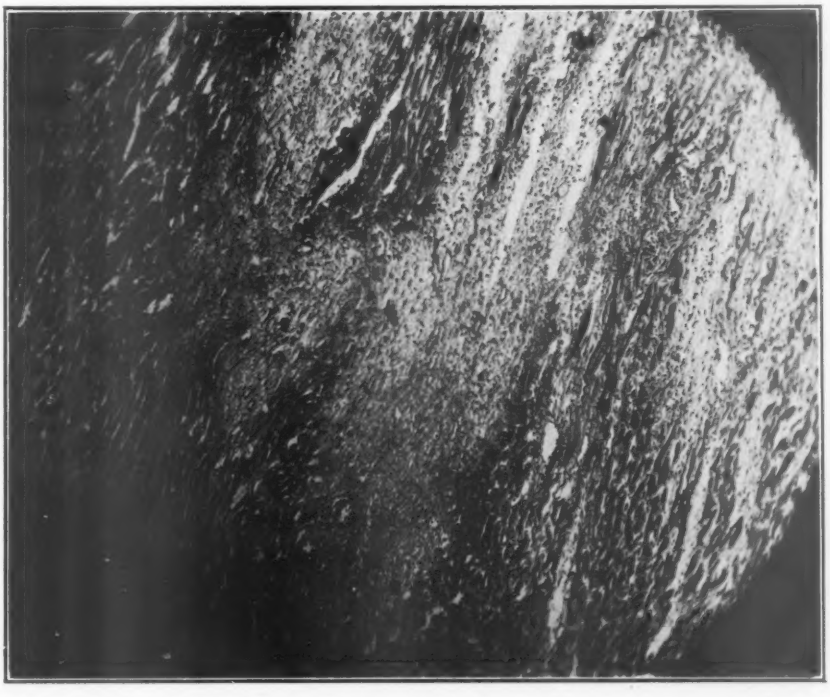

Fig. 1.—Low power section from the myocarditic area in the left ventricle, showing fibrous myocarditis produced by caffeine and adrenalin.

Twenty-four hours after injection the changes are not significant. The interstitial edema seems to be slightly more marked and the cross striations of the fibers less distinct.

Forty-eight hours after injection the interstitial edema is still slight. The muscle fibers appear thicker than normal and measure on the average 13.6 μ, the average width of the normal fibers being 7 to 10 μ. The nuclei are also enlarged and measure 0.45 by 4.2 μ as compared with 0.10 by 3.5 μ in normal hearts. The cross striations are even less distinct than after twenty-four hours. At this time an early proliferation of connective-tissue cells is seen which is most marked in the subendocardial and subepicardial regions and also around the

blood vessels, which latter often show considerable distortion in their course due to the contraction and pressure of the connective tissue about them; this condition is seen especially in the capillaries.

Four days after injection, the myocardial changes are more advanced. The edema between the muscle fibers is plainly visible. The muscle fibers are distinctly increased in size and show degenerative changes in many cases. They vary in different muscle fibers from loss of cross striations to solution of the fibers; some muscle fibers are vacuolated. The nuclei of the fibers remain preserved, but they are surrounded by a light nonstaining area, and are enlarged; furthermore, the occurrence of double nuclei is more common. The interstitial proliferation of connective-tissue cells is more pronounced.

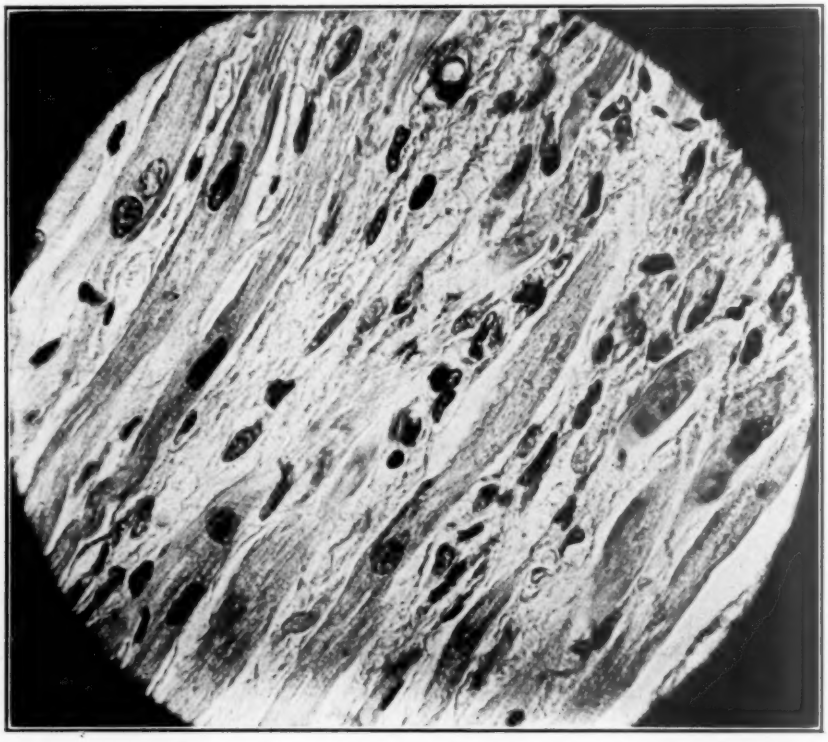

Fig. 2.—High power section of the myocarditis produced by caffeine and adrenalin, showing diffuse increase in interstitial tissue, with replacement of muscle fibers by fibroblasts, slight interstitial edema, degenerative changes in some muscle fibers, double nuclei, and distortion of the lumen of a blood capillary.

Six days after injection, the enlarged muscle fibers are separated by edema. The degenerative changes in these fibers are more advanced; some fibers are granular and a few are vacuolated. Double nuclei are frequently found. However, the most striking change at this time is the connective-tissue proliferation which originates in the connective tissue of the subendocardial, the subpericardial, and the perivascular areas.

Nine days after injection, the edema is quite marked. The muscle fibers again are found increased in size and there is also an increased

number of double nuclei. Degenerative changes, such as granulation, vacuolization and loss of cross striations are more pronounced. Some fibers are dissolved. The nuclei take a pale stain. The connective-tissue proliferation has increased and young connective-tissue cells are now seen extending deeply between the muscle fibers. This connective-tissue proliferation is most noticeable in the papillary muscles of the left ventricle.

Twelve and twenty-one days after injection, there is found only a little variation in the character of the lesions and these may consequently be described together. The interstitial edema is very marked; the degenerative changes, previously mentioned, are more pronounced; in particular, lysis of the muscle fibers is quite frequent. The fibers average from 16 to 18 μ in breadth as compared with from 7 to 10 μ in the normal heart. Vacuoles in the cells are very frequent and often they are multiple. The nuclei are larger than normal and by counting it was found that double nuclei average about 50 in 20 microscopic fields as compared with about 10 in 20 fields in the normal. Some muscle fibers are atrophic and consequently show an increase in brown perinuclear pigmentation and also a granular appearance and vacuolization. By staining with Sudan III we found these vacuoles usually to be due to the presence of fat droplets. The connective-tissue proliferation, although more diffuse at this stage, is most marked about the blood vessels and near the endocardium and pericardium. Christian[3] has shown that when the proliferation of connective tissue encroaches upon the endocardium at this point a proliferation of the latter occurs. This connective-tissue proliferation may also appear in the form of an organizing pericarditis. There is in addition a connective-tissue proliferation between the muscle fibers, which by contraction and pressure causes the blood capillaries in this situation to be constricted in some places and ballooned out in others, so that the course and the shape of the capillaries show considerable distortion. It may be that the frequency of abscess formation in these scarred areas, when bacteria are injected into the blood stream,[4] may be partly accounted for by this distortion of the capillaries, since bacterial emboli probably lodge more readily and in greater numbers in such capillaries than in those in which the lumina are regular and even.

Six weeks after injection, the edema between the muscle fibers has become less marked, thus indicating that absorption of the fluid has begun; the hypertrophy and degenerative changes in the muscle fibers are, however, still apparent, although to a less degree than at an earlier stage. Double nuclei are likewise quite frequent, and again some cells are vacuolated. The connective-tissue proliferation is even more extensive than before; it is found rather diffusely in the left auricle and ventricle between the muscle fibers as well as subpericardially and subendocardially. The papillary muscles, especially those of the left ventricle, are extensively fibrosed.

Eight weeks after injection, the edema has disappeared and the hypertrophy of the muscle fibers is now less marked. The degenerative changes, such as granulation and vacuolization are likewise considerably less pronounced, although not entirely absent. There are a few double nuclei. The connective tissue is still diffuse.

Ten to fifteen weeks after injection, edema is lacking and hypertrophy is diminished. Degenerative changes are practically absent. However, occasional double nuclei are seen and the connective-tissue proliferation is now the only marked change noticeable.

Twenty weeks after injection, the edema, hypertrophy, and degenerative changes in the muscle fibers have disappeared. The connective tissue now is the only pathological feature. However, the greater part of the proliferated connective tissue has become fibrous.

Fatty degeneration.—Sections of the myocardium of the left ventricle of the heart of 13 rabbits were stained for fat with Sudan III. These rabbits had received caffeine and adrenalin injections during periods varying between fourteen and ninety days previously. Ten of these animals showed fat to be present in the muscle fibers; in two additional rabbits there was a slight but definitely positive reaction, and only in one the result was negative. In ten normal hearts of rabbits no fat was found in the myocardium. The fat was present in droplets in the muscle fibers, usually around the edge of the myocardial lesion and in the degenerating cells inside the involved area. The muscle tissue of the right ventricle was free from fat, even in cases in which the left ventricular muscle showed extensive fatty degeneration.

CHANGES IN THE PERICARDIUM

Christian[3] has described a fibrous pericarditis which follows the injection of adrenalin and spartein. He has termed this a sterile, congestive pericarditis. In our series, in rabbits which die of acute edema of the lungs, following the injection of caffeine and adrenalin, the blood vessels under the pericardium of the left ventricle and left auricle are found markedly congested; occasionally we may notice with the naked eye, in a rabbit which dies at an early period following the injection, a thin exudate of fibrin covering the surface of the heart. In the course of time this fibrin becomes organized by granulation tissue. We have been able to find the lesion described by Christian, after use of adrenalin and spartein, also in animals injected with caffeine and adrenalin. Furthermore, we have observed subpericardial hemorrhages in several cases following the injection of the latter substances. However, in only two rabbits out of 39, which were injected with caffeine and adrenalin and examined for pericarditis, were we able to see a fibrinous pericarditis in the gross; but in 12 of these cases we found areas of organizing fibrinous pericarditis microscopically. These rabbits were examined at periods, varying between three and eighty days following the injections. Cultures of the

pericardial sacs were made in six of these cases; they were negative. Rabbits which had bronchopneumonia were excluded from this series, since bronchopneumonia very frequently causes a pericarditis, probably by direct extension of the pneumonic process through the pleurae into the pericardium. Such a combination of pneumonia and pericarditis, we observed in nine rabbits of this series. On the other hand, we have observed serosanguinous and fibrinous as well as chronic fibrous pericarditis in stock animals which have died of bronchopneumonia. Such changes must, therefore, not be attributed to the injection of caffeine and adrenalin.

ENDOCARDITIS

A proliferation of the endothelium immediately adjacent to the myocarditic area that follows the injection of spartein and adrenalin was first observed by Christian and his coworkers. The lesion is a microscopic one. Christian regarded this as a very constant change occurring in the majority of the rabbits which he had injected with caffeine and spartein. It appears that this proliferation of the endothelium takes place whenever the myocarditic alteration encroaches upon the underlying endothelium.

In our own observations, mural thrombi were not observed in any of our rabbits. However. we have seen in three instances, in rabbits which died immediately following the injections of caffeine and adrenalin, hemorrhages into the endocardium, particularly into the mitral valve. What bearing this may have upon the question of localization of bacteria at this site is problematical. It seems to us probable that these hemorrhages occur very frequently, since the cases in which they were observed by us occurred in sequence and were late in our series. It is possible that in other animals they were overlooked by us.

We shall describe in a subsequent paper[4] the effects of these lesions of the myocardium, endocardium, and pericardium upon the localization of abscesses in the heart following intravenous injection of a Staphylococcus aureus culture.

SUMMARY OF MICROSCOPIC LESIONS

The principal microscopic changes that resulted from a single intravenous injection of caffeine and adrenalin in the rabbits were:

1. Interstitial edema.
2. Hypertrophy of the muscle fibers and nuclei.
3. Degenerative changes in the muscle fibers, which vary in severity from loss of cross striations, combined with an increase in intensity of the longitudinal striations, to development of a granular condition, vacuolization and solution of the muscle fibers.
4. Connective-tissue proliferation between the muscle fibers origi-

nating in the subpericardial and subendocardial areas, and around the blood vessels.

5. Endocardial proliferation, or hemorrhages into the endocardium, especially in the mitral valve.

6. Pericarditis.

7. Fatty degeneration of the myocardial cells in and around the myocarditic area.

The edema and hypertrophy of the muscle fibers and the fatty degenerative changes are most marked from twelve to twenty-one days after the injections. This is probably the reason why the gross myocarditic lesion is most marked at this period. The disappearance of the gross lesions corresponds to the time when the microscopical changes, consisting in edema, hypertrophy, and the fatty degeneration of the muscle fibers, likewise disappear. The gross lesion is practically never seen later than the twentieth week following the injection, although some connective tissue is present at this time microscopically. It is perhaps well to emphasize here the complete absence of any leucocytic reaction in the myocardium.

CONCLUSIONS

1. One single injection of 0.025 gm. caffeine sodium benzoate with 0.2 c.c. of adrenalin causes myocarditic lesions in practically all of the rabbits.

2. The most frequent site of this lesion is in the papillary muscles of the left ventricle. Next in frequency are the myocardium of the left ventricle at the upper half of the anterior aspect, which in some cases encroaches upon the myocardium of the left auricle, then the upper half of the posterior aspect of the left ventricle.

3. The lesions appear a few days after the injections and consist of (a) interstitial edema; (b) hypertrophy of the muscle fibers and nuclei; (c) degenerative changes in the muscle fibers, which vary in severity from loss of cross striations and increase in intensity of the longitudinal striations, to granulation, vacuolization and solution of the muscle fibers; (d) connective-tissue proliferation; (e) proliferation of the endocardium adjacent to the myocardial lesion and hemorrhages into the endocardium; (f) pericarditis, and (g) fatty degeneration of the myocardial cells in and immediately around the myocarditic lesion.

4. The lesions are most marked from twelve to twenty-one days after the injections and are progressively less evident both grossly and microscopically at later periods.

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CHEST CLINICS IN IOWA

Occasional Heart and Lung Clinics in a Rural State

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THE combined heart and lung clinics, so called "chest" clinics, or clinical conferences, are a form of diagnostic and consulting service being offered to the physicians of Iowa by the Iowa Tuberculosis Association and the Iowa Heart Association, during the past two years. It is our purpose to explain the methods employed in arranging and conducting these clinics, to discuss certain of the related public health aspects, and to make a clinical report of the patients examined. So far as we can learn, no other state has as yet conducted clinics in the same manner. Missouri is preparing to do so in a somewhat similar way. Wisconsin and Maryland and other states also have held combined chest clinics.

In May, 1925, the Iowa Heart Association was formed, and shortly thereafter it was affiliated with the Iowa Tuberculosis Association, permission having been granted to the latter organization by the National Tuberculosis Association. The affiliation of these two agencies has been mutually helpful, as we shall point out later. The Iowa Tuberculosis Association had for several years held occasional clinics on lung diseases. Since the heart work has been added, the clinics include both lung and heart examinations, an examiner for each being in attendance.

Heart disease and tuberculosis are similar in certain respects. They frequently have their beginning in childhood; they develop slowly; they have symptoms in common; both may lie dormant for many years; usually they reach their climax in the productive years of life, and they require similar methods of treatment. Because these diseases exhibit such parallelism and because the case-finding methods and the conduct of the tuberculosis clinics can readily be adapted to serve patients with heart disease with but little added expense and effort, the combined chest clinic idea seems to be a sensible one and is worthy of trial wherever local conditions will allow it. Occasionally a request is received for a lung clinic alone or for a separate heart clinic, but it is the policy of the Association to give the two together.

The chief object of the clinic is to offer physicians in their own communities assistance in the diagnosis and treatment of patients with heart and lung diseases. For this reason we hold demonstration clinics, examining but a limited number of patients, rather than crowded

diagnostic clinics as formerly. Realizing the necessity of maintaining the integrity of the County Medical Society, and with the conviction that if any cooperative movement among physicians is ultimately to succeed, it must be done in and through the organized medical forces, we endeavor so to conduct the clinics that they shall at all times be in harmony with the wishes of the county societies.

The publicity attending the clinic offers a splendid means of disseminating popular information regarding heart and lung diseases. Public talks are often given by the clinicians to high-school assemblies and parent-teacher associations. The attention of the general public is called to the importance of periodic health examinations by the family physician. The clinic discovers early cases, suspects, and contacts; emphasizes the importance of childhood infection, and gives a fine opportunity to advise patients how to live so as to avoid future breaks in health. A distinctly valuable function of these clinics lies in the point that persons fostering the local public health or the Christmas seal associations in the county find an interest in the clinic, for their funds are used to pay the local share of the expenses. Thus, the home doctors and the members of the local lay health organizations are bound together in a common endeavor, often with the result that the two groups work in closer harmony.

In Iowa, before the establishment of the combined chest clinics, the campaign against tuberculosis had failed to arouse sufficient interest in certain counties to cause them to request lung clinics. Since the addition of the heart clinics, fifteen additional counties have held the combined chest clinics. Not all of the credit for this awakening of interest is due to the new appeal of the campaign against heart disease, for an intensive effort to organize these counties has recently been carried on by the Iowa Tuberculosis Association, but it may reasonably be assumed that the interest in the tuberculosis movement in some counties has been stimulated by reason of the local and nation-wide campaign against heart disease. Not infrequently during the examination of patients in the clinics the examiner in tuberculosis finds a patient whose symptoms or findings suggest heart disease. Such a patient is then referred to the examiner in heart diseases, thus offering the opportunity for two opinions. Not only the patient but also the clinicians and the patient's physician are benefited by this interchange of opinions. Compilation of careful statistics from such clinics may be the only means of obtaining state-wide information regarding the types and morbidity of heart and lung diseases.

The details of arrangement for the conduct of these chest clinics have been given a great deal of study and attention. Endorsement of the plan has been given by the Iowa State Medical Society, the Board of Control of State Institutions, and the State Board of Health. Clinics are held only upon written request from county medical societies,

such invitations being signed by the president or secretary of the society. In addition to this, a written request is expected from the local public health association or seal sale unit. Then, under the combined auspices of these agencies, the clinics are scheduled for some future date. Such a plan overcomes the objection raised by some physicians that clinics are sometimes conducted wholly under lay control without regard to the wishes of the physicians in the community. Upon receipt of the specified invitations, the Iowa Tuberculosis Association arranges with the custodian of the local seal sale funds to pay a part of the expenses and to make the necessary arrangements as to the place for the clinic and other details. There is no expense to the physicians individually or to the county medical societies.

Preliminary publicity is secured through the county newspapers by advance copy containing information relative to tuberculosis and heart diseases, together with notices about the time and place of the clinic. Reference is made in these newspaper articles to the fact that only a limited number of persons can be examined and that all those who desire to come must first consult their family physician, for only through him are patients scheduled for examination. It is found that the average county editor is liberal with his space for this purpose.

The conferences or clinics are held on an average of once a week, usually on Fridays. About one week before the date selected, the staff field nurse goes into the county to complete the arrangements with the local committee. She visits every doctor in the county, explaining the plan to those not familiar with it, and urging each doctor to select a patient or two for the clinic. Often she visits the patients to obtain preliminary histories. From the records of previous clinics in the county she makes a check on patients who have been examined before, those persons residing there who have been discharged from sanatoria for the tuberculous in the state, and those ex-service men who have had a diagnosis of tuberculosis. Other sources of information are used to discover every possible tuberculous individual in the county. The nurse also makes arrangements for talks to be given by the examiners to lay groups and to the physicians at a noon luncheon. Should the clinic be held in the fall, those who are directing the seal sale often use the clinic with its attendant relations to public health as a means of promoting the seal sale.

A few points regarding the actual conduct of the clinics may be mentioned. The physicians are invited to come with their patients and to present case histories and, if possible, laboratory reports. About one-half hour is allowed for each examination and an open clinical discussion follows. Certain instruments of precision have not been used thus far, but it is hoped that a portable electrocardiograph may be secured later to be used for demonstration purposes. If the

examiner finds that the patient has definite or suspected lung or heart disease, or if a potential cardiac patient is found, he makes it a point to urge upon the patient the advisability of periodic examination by his family physician. Patients diagnosed as having active pulmonary tuberculosis are usually urged to apply for treatment in the state or county sanatoria in Iowa. Unfortunately no adequate provision is made in this state for the care of convalescent patients with heart disease. This is greatly needed. After the completion of the examination, the examiner writes his opinion with suggestions as to treatment, the original copy of which is given to the referring physician or sent to him by mail, while the duplicate remains with

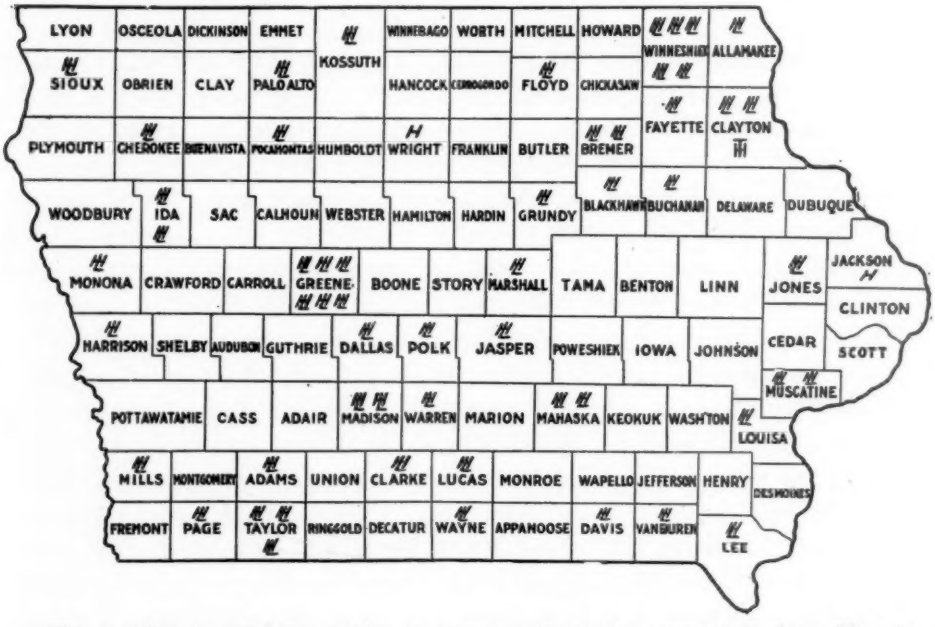

Fig. 1.—Heart and lung clinics in Iowa. The period covered is from May 21, 1925, to May 21, 1927. The symbol *HL* indicates combined heart and lung clinics. *H* indicates heart clinic alone.

the history. The physicians appreciate these reports. The examinations usually begin at nine A.M. and continue until five P.M., thus allowing time to examine ten or twelve patients in each section of the clinic. Occasionally, instead of devoting time to clinical discussion of patients during the forenoon, a few selected patients are presented at a clinical conference with the entire group of physicians in the afternoon.

The response on the part of the local physicians has been more than satisfactory as shown by an average attendance of from 50 to 100 per cent of the membership of the county medical societies. The interest varies in different counties dependent upon many factors, such as the attitude of the community toward public health work, the activities of one or more interested physicians, and the character

of the preclinic preparation by the staff nurse in charge. Reference is made to the map (Fig. 1), which shows the state-wide distribution of the clinics covered in this report and the number held in each county.

REPORT ON HEART CLINICS

The system of cardiac diagnosis used in the clinics has been described by Dr. Paul D. White.[1] The nomenclature adopted by the American Heart Association[2] is similar to the above mentioned and is preferable. Such classifications offer a satisfactory means of studying patients from the etiological standpoint, and are also helpful in the clinical teaching of heart disease. Because of the lack of time to make complete examinations, errors in diagnosis are bound to occur, but by rechecking the patients at subsequent clinics some such mistakes are corrected. The only instruments used in the examinations are the stethoscope and the sphygmomanometer, for it is felt that the general practitioner is able to make a correct diagnosis in at least 75 per cent of cases with these instruments alone, if he also takes a complete history and examines his patients with care.

TABLE I

CLASSIFICATION OF DIAGNOSES

	TOTAL NUMBER	PER CENT OF ALL PATIENTS	PER CENT OF HEART PATIENTS
Patients With Heart Disease (Etiological Types):			
Rheumatic	100	20.9	37.9
Arteriosclerotic	37	7.7	14.0
Hypertensive	32	6.7	12.1
Congenital	20	4.2	7.6
Angina Pectoris	20	4.2	7.6
Arteriosclerotic and Hypertensive	18	3.7	6.8
Uncertain Etiology	15	3.1	5.6
Syphilitic	9	1.9	3.4
Subacute Bacterial Endocarditis	4	0.8	1.5
Thyroid	4	0.8	1.5
Coronary Occlusion	2	0.4	0.8
Heart in Anemia	2	0.4	0.8
Toxic	1	0.2	0.4
Total	264	55.1	100.0
Patients Without Definite Heart Disease:			
Possible Heart Disease	35	7.3	
Nervous Hearts including Effort Syndrome	18	3.8	
Potential Heart Disease	10	2.1	
Total	63	13.2	
"No Heart Disease Found"	152	31.7	
Grand Total	479	100.0	

The following physicians have conducted a total of seven clinics: Dr. W. L. Bierring, Des Moines; Dr. V. C. Graber, Iowa City; Dr. John

Russell, Des Moines, and Dr. L. R. Woodward, Mason City. This report covers 48 clinics conducted by Myers between May 21, 1925, and May 21, 1927.

Table I represents the examination of 462 individuals. In 17 instances patients were listed twice according to the etiological types of heart disease; hence, there are 479 diagnoses for consideration. Heart disease was diagnosed 264 times in the 479 cases, and in 215 instances evidence of definite heart disease was lacking. The more common disorders of the heart in the order of their frequency in this series are: rheumatic, arteriosclerotic, hypertensive, congenital, angina pectoris, and arteriosclerotic and hypertensive. The rheumatic form occurs more than twice as frequently as the next in order, the arteriosclerotic. This is partly explained by the fact that the physicians are especially requested to send young people and children with suspected heart disease to the clinics.

Important Criteria for Diagnosis

Rheumatic Heart Disease.—The term "rheumatic heart" is not entirely satisfactory, but at present, it is used to cover practically all cases of nonsyphilitic valvular disease in persons under forty years of age, except congenital changes.

TABLE II

STRUCTURAL CHANGES IN 100 PATIENTS WITH RHEUMATIC HEART DISEASE

	NUMBER	PER CENT
Mitral Stenosis	41	41
Mitral Regurgitation	39	39
Mitral Involvement*	33	33
Aortic Stenosis	7	7
Aortic Regurgitation	42	42
Enlargement	70	70
Acute Pericarditis	1	1
Chronic Adhesive Pericarditis	2	2

*This condition occurred in combination with aortic stenosis or aortic regurgitation.

Table II shows the structural changes in the 100 patients with rheumatic hearts. There were 43 males and 57 females. Seventy-four were considered to have inactive, 16 active cardiac infection, and in 10 activity was doubtful. In 10 instances there was no demonstrable enlargement of the heart, in 7 enlargement was questionable, and in 13 there was no report. Symptoms or signs of congestive heart failure were present in 29 patients. The following cardiac arrhythmias were noted: auricular fibrillation, 16 times; premature contractions, 9 times; paroxysmal tachycardia, twice; probable sino-auricular block, once; possible partial auriculoventricular block, once. Sixty-seven had regular rhythm or simple sinus arrhythmia, and the rhythm is not

TABLE III

RHEUMATIC HEART DISEASE, ACCORDING TO AGE

AGES	NUMBER	PER CENT
1- 9	12	12
10-19	35	35
20-29	15	15
30-39	11	11
40-49	16	16
50-59	7	7
60-69	4	4
Total	100	100

reported in 4 cases. The functional classification as recommended by the American Heart Association was used: Class I, 25; Class IIa, 38; Class IIb, 22; Class III, 12; class not reported, 3.

Table III shows a separation of the cases of rheumatic heart disease according to age. The youngest was five, and the oldest was sixty-nine years of age. The highest per cent occurred from the ages of ten to nineteen. Wyckoff and Lingg[3] have compiled similar figures from three different observers, and in each instance the highest per cent of cases occurred during the age period from twenty to twenty-nine.

With limited time for examination one must dispense with many routine questions in the history, but must find out definitely regarding the occurrence of rheumatic fever, chorea, recurrent tonsillitis, growing pains, and scarlet fever. These are the most important etiological infections, particularly rheumatic fever and chorea. Symptoms play a lesser rôle in early life than in later life. Mitral stenosis is the most characteristic valvular disease in the rheumatic heart, therefore, its detection is most important. We try to elicit the low-pitched, mid-diastolic rumble, or the crescendo presystolic murmur at the apex, pathognomonic of mitral stenosis, by examining the patient in the upright, recumbent, and left lateral positions. Occasionally it is found necessary to increase the heart rate by exercise or by amyl nitrite to bring out these murmurs. If either of the murmurs is distinctly heard, the diagnosis of rheumatic heart disease is established, for it is felt that in this part of the country there is no other known cause for organic mitral narrowing than rheumatic disease. It is recognized that when aortic insufficiency is present, the other murmurs, those at the apex in particular, are sometimes difficult to interpret. To avoid possible error in the case where the etiology is not clear, though the findings are typical, it has been pointed out that one should qualify his diagnosis by calling the condition one of rheumatic *type* rather than definite rheumatic heart disease. The diagnosis of mitral stenosis was made in 41 per cent of the patients with rheumatic hearts. "Probable" rheumatic heart disease is diagnosed when there

is a definite history of rheumatic fever or chorea, if the patient has a very loud or long systolic murmur, with or without cardiac enlargement. Occasionally one finds aortic insufficiency to be the only detectable valve impairment in the rheumatic heart; usually, however, it is associated with mitral stenosis. Aortic insufficiency was diagnosed 62 times in the 264 cases and was considered to be due to rheumatic involvement in 42 cases, syphilitic in 8, possible hypertensive and arteriosclerotic heart disease in 1, arteriosclerotic in 1, and 10 cases were of uncertain origin, of which 7 were probably rheumatic. The diagnosis of "mitral involvement" or "mitral impairment" has been made a number of times in young persons with a clear history of rheumatism or chorea, who have loud apical systolic murmurs, but without definite evidence of mitral stenosis, with or without aortic stenosis or regurgitation. Aortic stenosis is considered to be rheumatic in origin in most instances, though its exact diagnosis is often difficult. Tricuspid or pulmonic valve disease was not found in the rheumatic hearts. Cardiac enlargement in young persons signifies heart impairment. Auricular fibrillation in individuals under fifty years of age suggests a rheumatic etiology. Acute pericarditis and chronic adhesive pericarditis, both rare in this series, usually result from rheumatic infection. The latter condition is very difficult of diagnosis. Because of the great importance of rheumatic heart disease to the general practitioner, much time is spent in discussion of the etiology, prevention, and treatment.

Arteriosclerotic Heart Disease.—One should suspect this type in persons over fifty years of age who present any of the following symptoms or signs: breathlessness on exertion, edema of feet or ankles, auricular fibrillation, anginal attacks. Hypertension and peripheral arteriosclerosis are not expected to be constant findings in this condition.

Hypertensive Heart Disease.—This type results from either "essential" hypertension or nephritis. The family physician wants to know the earliest signs of heart involvement in persons with hypertension and, though it is often difficult to determine them, such symptoms as breathlessness on exertion, nocturnal dyspnea, substernal or precordial pain or aching, forcible palpitation, suggest beginning myocardial weakness. Eventually in such cases there follows cardiac enlargement, congestive failure, angina pectoris, vascular accident, or renal insufficiency.

Congenital Heart Disease.—History of patients who have been "blue babies," persistent cyanosis of extremities and face, clubbing of fingers and toes, structural changes in the heart, usually associated with this type of cardiac disease, are important diagnostic points. Congenital heart disease was found in patients who had no evidence of such

condition except the anatomical changes. The high proportion of congenital heart disease in this series is probably explained by the fact that physicians bring such patients to the clinic on account of their rarity and unusual interest.

Angina Pectoris.—The criteria for the diagnosis of angina pectoris rest largely upon the history of pain, constriction, heaviness, or other form of distress in the substernal or precordial region, with or without radiation, the distress being precipitated by exertion and relieved by rest and nitrites. It is rarely present in persons under fifty years of age. In discussing this condition, the examiner has an opportunity to emphasize the importance of pain.

Arteriosclerotic and Hypertensive Heart Disease.—When a patient has definite hypertension with well-marked arteriosclerosis and especially if auricular fibrillation is present, he is placed in the arteriosclerotic and hypertensive group.

Uncertain Etiology.—This class includes for the most part adults with diastolic murmurs along the left sternal border not associated with other valve disease, where differentiation between syphilitic and rheumatic or other form of etiology is difficult; children with loud murmurs not clearly rheumatic or congenital in origin; and persons with enlargement, little evidence of valve disease and slight hypertension when one must consider the hypertensive, the thyroid, and the arteriosclerotic form.

Syphilitic Heart Disease.—The diagnosis of cardiovascular syphilis is seldom made until the heart muscle weakens, producing symptoms, or the aortitis becomes extensive enough to involve the aortic ring, producing aortic insufficiency. Lues is always to be suspected in males over forty years of age who have symptoms such as breathlessness, chest discomfort, or nocturnal dyspnea of recent origin. Since aortic regurgitation is an early sign of the disease, the examiner devotes considerable time to discussing important features about the diastolic murmur, especially the soft, high-pitched quality and the fact that it is frequently heard most clearly along the left sternal border.

Bacterial Endocarditis.—Only the subacute form was found in 4 patients. It is often overlooked as a complication of rheumatic heart disease. Common clinical evidences are: protracted fever, weakness, visceral embolic phenomena, skin manifestations, Osler's nodes, and enlargement of the spleen. Blood cultures are often needed to establish the diagnosis.

Thyroid Heart Disease.—This is a relatively uncommon form in our experience. One should make the clinical diagnosis of this condition only when there is evidence of more abnormality than simple tachycardia, like permanent or paroxysmal auricular fibrillation, cardiac

enlargement, or congestive failure. The clinician finds it difficult to determine the point of change from a normal to a diseased heart in hyperthyroidism. Occasionally one finds cardiac enlargement to be the only discoverable structural change in the heart which, in the absence of hypertension, arteriosclerosis, or signs of chronic adhesive pericarditis, suggests chronic hyperthyroidism as the cause of the enlargement. Early diagnosis and prompt treatment have reduced the number of thyroid hearts.

Coronary Occlusion.—Two of the patients with angina pectoris gave clear histories of attacks simulating coronary occlusion previous to our examination at the clinics. Their histories were characteristic: severe protracted chest pain, requiring morphine for relief, associated with marked prostration and a long period of convalescence. Other details of the diagnosis of coronary occlusion will not be discussed here. Some practitioners do not as yet distinguish between angina pectoris and coronary occlusion.

The Heart in Anemia.—Two patients with evident anemia were thought to have heart muscle weakness and systolic murmurs complicating the anemia.

Toxic Heart.—Rarely one sees persons with gall bladder disease with cardiac signs or symptoms which seem to result therefrom. Excessive tobacco smoking or coffee may make the heart irritable.

No diagnosis of diphtheritic heart disease was made. None of the rare infectious types, like that from tuberculosis, was found, and in no instance did we make the diagnosis of "athlete's" heart, the "beer" heart, or "fatty" heart. No case of cardiac tumor was discovered. A considerable number of patients, 31.7 per cent, failed to show positive evidence of heart disease.

For the purpose of further study, some patients must be placed in the "possible heart disease" class. They usually have abnormal signs and symptoms, like paroxysmal auricular fibrillation, with no other evidence of cardiac impairment, apical systolic murmurs which are difficult to interpret, or hyperthyroidism or hypertension with cardiac symptoms of indefinite nature.

With the nervous heart is included effort syndrome. The diagnosis of nervous heart is being made by us less frequently than formerly, especially in clinics where only comparatively short periods of observation are permitted. To tell one that he has a nervous heart often only adds fuel to his mental fire. Such patients are usually placed in the "no heart disease found" group.

Potential heart disease patients are for the most part young persons with definite history of rheumatic fever or chorea, in whom there is no proof of heart disease at the time of examination.

REPORT OF LUNG CLINICS

The method of diagnosis of pulmonary tuberculosis in use in the lung clinics is based upon case analysis through study of the cardinal points, according to Dr. Lawrason Brown,[4] of Saranac Lake. His original statement is as follows: "After much study, five essential diagnostic data have been selected at Trudeau Sanatorium: (1) a history of hemoptysis, (2) presence of pleurisy with effusion, (3) the presence of tubercle bacilli, (4) the occurrence of moderately coarse râles above the third rib and third vertebral spine, and (5) the occurrence of diffuse mottling (parenchymatous lesion) in the same area on the x-ray plate. The absence of all five of these data demands a negative diagnosis; the presence of either the first or second datum makes a diagnosis of suspected tuberculosis advisable."

In practical use, therefore, great dependence must be placed upon an unexplained history of wet pleurisy or hemoptysis, and upon careful auscultation of the chest for posttussic apical râles in making a diagnosis. Occasionally an x-ray film is available or obtainable, though the average film is of doubtful value, especially in early cases. Sometimes the sputum has been examined, usually not at all, rarely more than once.

With these diagnostic limitations clearly in mind, we feel that a brief report of the result of the examinations for lung diseases at our clinics may be helpful to others. From May 21, 1925, to May 21, 1927, 55 combined chest clinics were held in various counties in the state. Two clinics were held by Dr. Harold E. Johnson, of Des Moines; two by Dr. J. Carl Painter, Superintendent of Sunny Crest Sanatorium at Dubuque; one by Dr. O. W. Britt, Assistant Superintendent of the Sanatorium at Worthington, Minnesota; and five by Dr. H. V. Scarborough, Superintendent of the State Sanatorium at Oakdale. The remaining 45 clinics were conducted by one of us (Peck). It seems preferable that this latter number only be considered in the following statistical study.

TABLE IV

AGE GROUPS OF PATIENTS EXAMINED

Adults	462	85.1 per cent
Children	81	14.9 per cent
Total	543	100.0 per cent

All patients over fourteen years of age were classified as adults, and those fourteen years or under were classed as children. The attendance of children at the lung clinics was discouraged during the past two years more than formerly. The reason for this rule is obvious; the impossibility of making a positive diagnosis of tuberculous infection in children without the use of the x-ray and tuberculin. It is very rare to find chronic ulcerative pulmonary tuberculosis before

the age of fifteen, there being but one instance in this series of 81 children. Some years ago we included a head surgeon and a pediatrist in our diagnostic group and then gave a more complete diagnostic service to children. These features, however, seemed to detract from what we regarded as our prime objective; that is, the recognition of definite lung diseases.

Separation of the 543 patients into the two natural divisions of tuberculous and nontuberculous, revealed the surprising fact that almost exactly one-half of them fell into each class.

TABLE V

PATHOLOGICAL CLASSIFICATION

Number of patients found tuberculous	273	50.3 per cent
Number of patients found nontuberculous	270	49.7 per cent
Total number of patients examined	543	100.0 per cent

The reader should again be warned that the refinements of diagnosis are lacking in these one-day clinics held in churches, court houses, schools, lodge halls, and hospitals, under adverse conditions. No doubt a more careful study over a longer period of observation would materially alter these figures.

The 273 patients in whom pulmonary tuberculosis was diagnosticated, or at least strongly suspected, may be further divided according to the clinical evidence of disease, as follows:

TABLE VI

CLINICAL CLASSIFICATION

	NUMBER	PER CENT
Pulmonary tuberculosis, active	146	53.5
Pulmonary tuberculosis, nonclinical	36	13.2
Tuberculosis "suspects"	70	25.6
Tuberculous adenitis	7	2.6
Chronic pleurisy, dry	11	4.0
Pleurisy with effusion	3	1.1
Total tuberculous patients	273	100.0

Exact estimation of activity in many instances presented an exceedingly difficult problem, necessarily to be solved almost instantaneously. Usually the clinical sense of the examiner was the determining factor, guided by the temperature, the pulse rate, the moisture on auscultation, and perhaps very largely by the invaluable information obtained from the family physician.

The group of inactive cases was made up principally of ex-sanatorium patients, together with a certain number showing unmistakable evidence of chronic fibroid phthisis. The examination of former sanatorium patients is regarded as an especially valuable form of follow-up work, as it is not feasible for many such patients to return to their

sanatorium for their periodic reexaminations, the necessity for which is always stressed. Complete files are maintained at the office of the Iowa Tuberculosis Association of admissions to and discharges from our public sanatoria, as well as other known active cases in the state, thus making the records available to our clinic nurses.

The "suspects" include many cases which are susceptible of more exact classification, with more time and better equipment. The attending physician is always urged to study carefully such patients, making use of the case analysis system previously described.

Tuberculous adenitis is doubtless much more common than we have shown here. In the seven cases mentioned the glandular involvement seemed to be the principal pathology. Some of these no doubt were hilus tuberculosis, but lacked the necessary x-ray and tuberculin confirmation.

The remaining 270 cases in which no tuberculosis could be determined presented an interesting group. In 201 of them no pulmonary pathology was found, leaving 69 cases of nontuberculous pulmonary infection which deserve more extended comment.

TABLE VII

NONTUBERCULOUS PULMONARY INFECTIONS

	NUMBER	PER CENT
Bronchitis, various types	18	26.1
Neoplasms	5	7.2
Chronic Empyema	5	7.2
Bronchial Asthma	13	18.9
Bronchiectasis	28	40.6
Total	69	100.0

The bronchitis cases, including acute, subacute, and chronic, of various types, deserve more careful differential diagnosis than our limited time permitted. More than likely repeated sputum examinations would materially reduce the number in this group. The importance of examinations of the sputum by a reliable technician was always impressed upon the general practitioner, so that possible diagnostic errors would be kept at the minimum.

Primary pulmonary neoplasm is believed to be much more common than is generally regarded. One per cent should not be too high a percentage to discover in such patients. Only one case was definitely proved at autopsy, but the others were clinically very suggestive.

Chronic empyema rarely becomes a causative factor in pulmonary tuberculosis, although a frequent source of worry to the patient. The five instances were all postoperative, in two cases with poor surgical results.

Bronchial asthma was diagnosticated in 13 instances, usually upon a clear history of paroxysmal attacks of spasmodic bronchitis or from the evidence submitted by the medical attendant. Typical musical

râles were heard in many cases. Obviously no further effort was made to analyze such patients as to their allergic phenomena, etc.

Bronchiectasis is apparently frequent in Iowa, since the influenza epidemic which, partially at least, accounts for the 28 cases seen. The diagnosis is usually not difficult, based upon the chronicity, the history, the character of the sputum, negative bacillary reports, and characteristic physical findings. Postural drainage is urgently advised in all such patients, especially the children, and beneficial results are frequently obtained. The use of iodized oil is routinely suggested in order to determine the exact extent and location of the pulmonary pathology and to differentiate this condition from lung abscess.

The group of 201 patients without evident tuberculosis was made up of a large number of contacts, a few neurotics, and many with extrapulmonary disease, such as nasal, dental, sinus, tonsillar, pharyngeal, cardiac, thyroid, intestinal, etc., even after careful selection of presumably suitable clinical material. This experience presents a strong argument for periodic health examinations.

An earnest effort was made to advise all the tuberculous patients carefully regarding their future care, as shown in Table VIII.

TABLE VIII

DISPOSITION OF TUBERCULOUS PATIENTS

	NUMBER	PER CENT
Advised sanatorium treatment	119	43.6
Advised close medical supervision	102	37.4
Advised home rest régime under physician	52	19.0
Total	273	100.0

With profound respect for institutional care of tuberculosis, we found 27, or nearly 20 per cent, of the patients with active pulmonary disease whom we failed to advise to go to a sanatorium. Some of these were elderly persons who do not adapt themselves to prolonged hospital régime; some were ex-sanatorium patients, who are faithfully carrying on after their institutional training; some had exceptional home conditions and an unstable nervous balance, who should have a trial at home under the care of a competent physician in whom they have implicit confidence; and a few were terminals who can be isolated, and whom the sanatorium cannot especially benefit. All patients with pleurisy with effusion out of a clear sky are considered tuberculous and advised to accept sanatorium treatment.

The inactive cases of closed tuberculosis present no hazard to others so long as they remain sputum free, but they rarely can be depended upon to continue under sufficiently close medical supervision.

The relatively large group of "suspects" cause us real trouble in diagnosis. Only exceptionally will they accept adequate medical observation, and they seem perfectly satisfied to neglect periodic reex-

aminations. Some form of legal persuasion for such patients would have our hearty endorsement. A large proportion of these suspects should have an intracutaneous tuberculin test, for the psychic value of an ocular demonstration of their tuberculous infection may produce a realization of their physical condition.

SUMMARY

1. Chest clinics as conducted in Iowa for two years, though still somewhat experimental, offer a valuable medical service in the campaign against tuberculosis and heart disease.

2. Organizations sponsoring the clinics find them mutually valuable in several ways:

a. They are a form of health work dealing specifically with diseases of the heart and lungs.

b. They furnish a means of carrying the message of health into the home communities.

c. The individuals conducting the seal sale observe tangible results from the use of their funds.

d. The talks to school children supplement the child health education program of the tuberculosis associations.

3. The clinics stimulate interest among physicians in public health work, thereby often bridging the gap between them and the local lay health organizations.

4. The clinical conferences and discussions of specific cases are apparently appreciated by the physicians.

5. A report is made here on the heart clinics which includes a classification of diagnoses, the structural changes found in patients with rheumatic hearts, and a discussion of the important criteria for diagnosis.

6. A report is also made on the lung clinics giving a pathological and clinical classification of all patients examined, according to the Trudeau scheme, and a résumé of certain diagnostic problems in lung diseases.

7. Should a similar plan of holding chest clinics be undertaken in several states, a standardization of the publicity, nomenclature, and clinical methods is advisable.

We desire to express our thanks to the staff of the Iowa Tuberculosis Association and the Iowa Heart Association, particularly to the Director of Clinics, Miss Lucy McMichael, R.N.

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HEART DISEASE AND DISORDERS IN NEW ENGLAND

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REALIZING the necessity of etiological studies before there can be a clear understanding of many cardiovascular problems, especially that of the prevention of heart disease, we began several years ago an investigation of the incidence of the various types of heart disease and disorders in New England. Since no single series of patients—private, hospital, or clinic—gives a correct incidence of community heart disease, we determined to include in our series a large number of cases from the several sources for comparison, as well as for a greater accuracy of the series as a whole. The entire group comprises 3000 cases, divided as follows: Group I, 1000 consecutive heart cases seen in consultation by one of us; Group II, 1000 heart cases seen at the Massachusetts General Hospital, 500 being ward patients, and 500 seen in the child and adult heart clinics of the Out-Patient Department; Group III, 1000 consecutive heart cases seen in general practice by thirteen reliable and representative general practitioners throughout New England. The patients sought advice for cardiac symptoms or signs or both. The diagnoses were clinical; a few were checked by autopsy.

Dublin[1] gives interesting statistics concerning organic heart disease, stressing the importance at the present time of carefully studied and followed series. He quotes in part the findings of Wyckoff and Lingg,[2] who later gave a detailed report of 1000 cases seen in New York. Wood, Jones, and Kimbrough[3] have given an interesting comparative study between groups studied in Virginia and Massachusetts.

In a recent publication, White[4] reviews the mortality statistics in Massachusetts. It is an interesting fact that though the death rate in Massachusetts has fallen from 2.15 per cent in 1875 to 1.23 per cent in 1925, the heart disease mortality has risen from 0.119 per cent in 1910 to 0.208 per cent in 1925. The author gives the following reasons for increase in deaths due to heart disease:

1. Diagnosis is more accurate, and fewer cases are overlooked.
2. Decrease in infant mortality and increase in duration of life allow more people to reach heart disease age.
3. It is likely that heart disease is actually on the increase in Massachusetts.

*Work carried out while a Dalton Scholar, Massachusetts General Hospital, Boston, 1925-26.

PRESENT OBSERVATIONS

The data assembled in the present study allow the analysis of many instructive details. The majority of the cases collected are from Massachusetts, but many are from various parts of New England, and the series is considered to be representative of New England as a whole. The classification used is like that of White and Myers[5] published in 1921, and revised in 1925,[6] and similar to that published by the American Heart Association.[7] The complete data may be re-

TABLE I

Group I. 1000 Cardiac Cases Seen in Consultation

Type	Sex		Total	Ages										Auricular Fibrillation	Paroxysmal	Block			Extrasystoles	Tachycardia	Angina Pectoris	Coronary Occlusion	Pericarditis	Valve Lesions													
	Male	Fem.		0-10	11-15	16-20	21-25	26-30	31-35	36-40	41-45	46-50	51+			Dis. A-V.	Org. A-V.	Intraven.	Bundle Branch	Bifurcation				A.R.	A.S.	A.R. + A.S.	M.R.	M.S.	M.R. + M.S.	A.R. + M.R.	A.R. + M.S.	Incompetent	A.R. + A.S. + M.R.	A.R. + A.S. + M.S.	A.R. + A.S. + M.R. + M.S.	Aort. Ins.	Aort. Ins. + Aort. Sten.
Rheumatic	74	83	157	14	27	26	32	32	20	6				35	3		4	2			75	2		13	13		1	26	31	17	14	13	2	3	25	9	1
Rheumatic + Arteriosclerotic	3	2	5						2	2	1			1	1			1				2			1	1										1
Rheumatic + Hypertensive	1	1	2					1		1				1	1													1	1							
Rheumatic + Art. + Hyper.	1	1	2						1	1				1							1							1						1		
Rheumatic + Subacute Bact. Endocarditis	6	3	9		5	2		1	1								1				3			3	1		7	7		2			5		1
Rheumatic + Effort Syndrome	14	11	25			5	11	9																					3	4					
Arteriosclerotic	125	37	162				1	6	35	84	36	16	17	1	13	11	3	5	54	31			3			17			3						
Hypertensive	23	29	52				1	16	18	15	2		2				3	2	3																
Arteriosclerotic + Hypertensive	59	36	95					3	19	50	23	14	5		4	6	8	1	20	8			5			6									
Hypertensive + Uremic	3	0	3		1	2											1	1																	
Hypertensive + Thyroid	0	2	2					1			1								1																
Hypertensive + Luetic	0	2	2					1	1														2												
Hypertensive + Effort Syndrome	3	10	13				1	5	6	1																									
Hypertensive + Luetic + Art.	1	0	1							1						1	1						1												
Hypertensive + Art. + Effort Syndrome	0	1	1						1																										
Arteriosclerotic + Luetic	2	0	2							1	1	1									1		2												
Arteriosclerotic + Emphysema	1	0	1							1																									
Arteriosclerotic + Effort Syndrome	2	5	7						3	4			1																						
Angina Pectoris	30	12	42			1		8	21	11	1							1	42				14												
Luetic (2 ♂ Aneurysm)	17	1	18					3	11	4											5	2													
Thyroid	0	3	3					3												1															
Myxoedema	0	1	1					1																											
Emphysema	1	4	5	1				1		3	1																								
Congenital	5	4	9	4		2	2	1															Entrance Septal Defect					Patent Duct Art.		Pulmonary Stenosis					
Congenital + Effort Syndrome	1	0	1			1																							1						
Subacute Bacterial Endocarditis	2	1	3				3																2						1						
Irritable Heart & Premature Beats	34	26	60		2	6	11	20	11	9	1		1																						
Irritable Heart & Paroxysmal Tachycardia	21	25	46		4	6	12	10	11	3										46															
Sinus Arrhythmia	1	1	2	1		1																													
Functional Systolic Murmurs	16	6	22	5	3	6	4	3	1																										
Effort Syndrome	87	120	207	1	13	48	38	57	24	6					1																				
Unknown	29	11	40		1	5	3	8	18	5		1	1	7		2					1	1	1			3									
Total in Numbers	562	438	1000	25	55	110	141	190	204	208	67	30	38	20	24	21	21	17	75	129	42	14	47	2	1	60	40	18	23	17	2	3	32	9	0	3 0
Total in Percentage	56.2	43.8	100	2.5	5.5	11	14.1	19	20.4	20.8	6.7	3	3.8	2.0	2.4	2.1	2.1	1.7	7.5	12.9	4.2	1.4	4.7	0.2	0.1	6	4	1.8	2.3	1.7	0.2	0.3	3.2	0.9	0	0.3 0

viewed by study of individual groups (Tables I, II, III), and the complete group as given in Table IV. The individual types of organic and functional disorders will be discussed in brief, with remarks concerning the variations in each group of cases and the completed group representing, we believe, the true community incidence of etiological factors. The fact is well known and yet often neglected that both private practice and public hospital practice must be included in a study of community disease. Neither one alone is representative.

GENERAL CONSIDERATIONS

The sex, age, and other broad divisions of the groups may be seen in Table V. There was found no predominance so far as sex was concerned. In the series, 59.4 per cent of the patients were above forty years of age, the largest single age group being from forty to sixty (33.3 per cent). The large number of patients prior to forty years of age resulted chiefly from the frequency in all series of the rheumatic type of heart disease, this being particularly prevalent in the hospital group, which represents the lower economic level of the community in which rheumatic infections are commonest.

TABLE II

Group II. 1000 Cardiac Cases {500 Cardiac Clinic Out-Patient Department Patients. 500 Hospitalized Patients.

Type	Sex: Male	Female	Total	Ages: 0-10	10-20	20-30	30-40	40-50	50-60	60-70	70+	Auricular Fibrillation: Permanent	Paroxysmal	Block: A-V Dig.	A-V Org.	Intraventr.	Auricular Flutter	Alternation	Paroxysmal Tachycardia	Angina Pectoris	Coronary Occlusion	Pericarditis
Rheumatic	206	220	426	67	188	51	54	39	22	5		57	6	4	11	1	2		10	2		40
Rheumatic + Arteriosclerotic	5	0	5						1	1	3	1			1	1				1		
Rheumatic + Hypertensive	5	2	7				4	1	2			1										1
Rheumatic + Luetic	2	0	2					2								1						
Rheumatic + Hypertensive + Luetic	0	1	1					1														
*Rheumatic + Congenital	3	1	4		4																	
Rheumatic + Subacute Bact. Endocarditis	8	10	18	1	8	5	4								1					1		3
Rheumatic + Effort Syndrome	3	8	11		2	3	4	2											1			
Arteriosclerotic	66	22	88					8	32	40	8	17	5	2	4	12				28	6	
Hypertensive	23	42	65			4	11	16	20	11	3	5	1			1			1	1		
Arteriosclerotic + Hypertensive	73	59	132		1			29	42	45	15	21	1		7	30	1	1	1	28		
Hypertensive + Thyroid	3	15	18		2	2	5	2	6	1		5							1			
Hypertensive + Luetic	2	2	4					1	3													
Hypertensive + Arteriosclerotic + Luetic	1	0	1							1												
Hypertensive + Emphysema	0	1	1					1														
Hypertensive + Effort Syndrome	0	2	2						2													
Arteriosclerotic + Luetic	1	0	1							1					1	1				1		
Arteriosclerotic + Emphysema	3	0	3					1			2									1		
Angina Pectoris	5	2	7					3	1	1	2									7		
Luetic (5 ē aneurysms)	27	8	35			4	8	7	10	6				1	1	2				13		
Thyroid	5	18	23		1	6	8	7	1			3	5		1							
Congenital	8	8	16	4	10	2									1				[illegible] Sept. Defect: 8			
Subacute Bacterial Endocarditis	2	0	2					2														
Effort Syndrome	13	22	35		4	11	12	7	1										1			
Irritable Heart ē Premature Beats	2	4	6	1			1	3		1												
Irritable Heart ē Paroxysmal Tachycardia	7	10	17		3	2	3	5	4										17			
Functional Systolic Murmurs	30	32	62	24	34	2		1	1			•										
Unknown	3	5	8	2		1	3	1		1		1			1		1					
Total in numbers	506	494	1000	99	257	93	117	139	148	114	33	111	18	7	29	29	4	1	32	83	6	44
Total in percentage	50.6	49.4	100	9.9	25.7	9.3	11.7	13.9	14.8	11.4	3.3	11.1	1.8	.7	2.9	2.9	.4	.1	3.2	8.3	0.6	4.4

Type	Valve Lesions: A.R.	A.S.	AR+AS	M.R.	M.S.	MR+MS	AR+MR	AR+MS	AR+AS+MR	AR+AS+MS	AR+MR+MS	AR+AS+MR+MS	AS+MR+MS	AS+MR	AS+MS
Rheumatic	11	1	2	29	65	150	4	46	1	3	86	19	4		1
Rheumatic + Arteriosclerotic		1						1				2			
Rheumatic + Hypertensive			1	1		2		1			1		1		
Rheumatic + Luetic					1			1							
Rheumatic + Hypertensive + Luetic															
*Rheumatic + Congenital				1		2					1				
Rheumatic + Subacute Bact. Endocarditis				3	1	7		2			4	1			
Rheumatic + Effort Syndrome				1	3	3		2			1		1		
Arteriosclerotic	3	1		5											
Hypertensive	1			1											
Arteriosclerotic + Hypertensive	7			6											
Hypertensive + Thyroid															
Hypertensive + Luetic	3														
Hypertensive + Arteriosclerotic + Luetic							1								
Hypertensive + Emphysema															
Hypertensive + Effort Syndrome															
Arteriosclerotic + Luetic	1														
Arteriosclerotic + Emphysema															
Angina Pectoris															
Luetic (5 ē aneurysms)	28														
Thyroid															
Congenital	Pulmonary Stenosis: 5							Patent Ductus Arteriosus: 3					Combinations: 4		
Subacute Bacterial Endocarditis							1				1				
Effort Syndrome															
Irritable Heart ē Premature Beats															
Irritable Heart ē Paroxysmal Tachycardia															
Functional Systolic Murmurs															
Unknown			1			1									
Total in numbers	54	3	4	47	70	165	6	53	1	3	94	22	6	0	1
Total in percentage	5.4	0.3	0.4	4.7	7	16.5	0.6	5.3	0.1	0.3	9.4	2.2	0.6	0	0.1

* Congenital Defect given under heading of Congenital Heart Disease

Because of variation in opinion concerning the mechanism of angina pectoris and auricular fibrillation, these disorders have been grouped alone when there was no other demonstrable cardiac abnormality, avoiding their inclusion in either the organic or functional groups. Over three-fourths of the completed group, 2,314 cases (77.1 per cent), constituted cases of organic heart disease, while the functional cases were about one-fifth of the total series, 579 cases (19.3 per cent). Uncomplicated angina pectoris (2.6 per cent) and uncomplicated auricular fibrillation (1.0 per cent) will be discussed under appropriate headings.

ORGANIC HEART DISEASE

Rheumatic.—By far the most conspicuous group, denoting a single etiological factor, was made up by the rheumatic type of cardiac involvement. Eight hundred and thirty-six cases, or 27.9 per cent, fell under this heading, while those cases exhibiting some complication along with the rheumatic disease, brought the number to 956 (31.9 per cent). Of the 2,314 (77.1 per cent) cases of organic heart disease in the series, 36.1 per cent were rheumatic in origin, while 5.2 per cent more were rheumatic with an added complication. Hence 41.3 per

TABLE III

Group III. 1000 Cardiac Cases Collected from General Practice.

Type	Sex			Ages								Auricular Fibrillation		Block							Valve Lesions													
	Male	Female	Total	0-10	10-20	20-30	30-40	40-50	50-60	60-70	70+	Permanent	Paroxysmal	Partial	Complete	Ventricular Alternation	Paroxysmal Tachycardia	Pericarditis	Angina Pectoris	Coronary Occlusion	A R	A.S.	AR+AS	M R	M S	MR+MS	AR+MR	AR+MS	AR+MS+MR	AR+A.Sy	MR+MS	AR+MR+MS	AS+MR	AS+MS
Rheumatic	83	170	253	33	63	39	48	34	21	14	1	50	7	1			2	8	3	1	4	4	3	79	48	71	11	1	8	7	1	1
Rheumatic + Arteriosclerotic	4	1	5						3	2		1							1			1				1							
Rheumatic + Hypertensive	2	5	7				2		3	2			1								1		1	1	1	2	1						
Rheumatic + Arteriosclerotic + Hypertensive	1	0	1							1									1						1								
Rheumatic + Thyroid	1	0	1				1																			1							
Rheumatic + Effort Syndrome	1	0	1				1																			1							
Rheumatic + Subacute Bact. Endocarditis	8	4	12	1	2	4	4	1										1							1	6	1	1					
Rheumatic + Emphysema	0	1	1						1																								
Rheumatic + Hypertensive + Toxic	0	1	1						1																1								
Arteriosclerotic	77	90	167					2	27	65	73	19			2	1	2	2	36	11	4	1		6									
Hypertensive	35	70	105		1	1	4	27	40	26	6	6	1				2	2	15	2	2												
Arteriosclerotic + Hypertensive	77	92	169					3	28	72	66	20	2		2		3	1	46	5	12			1									
Hypertensive + Uremic	5	5	10			1		3	5		1	2							1														
Hypertensive + Thyroid	1	0	1						1																								
Hypertensive + Luetic	4	0	4						2	2		2							1		4												
Hypertensive + Arteriosclerotic + Luetic	2	0	2						1	1							1		1		2												
Hypertensive + Arteriosclerotic + Uremic	3	1	4						1	1	2						1																
Arteriosclerotic + Luetic	1	0	1						1										1		1												
Arteriosclerotic + Uremic	2	2	4						3	1							2																
Arteriosclerotic + Emphysema	3	1	4						3	1			1						1														
Arteriosclerotic + Effort Syndrome	1	0	1							1																							
Angina Pectoris	14	14	28				2	4	16	5	1								28	1													
Luetic (+ Aneurysms)	18	3	21	1			3	10	5	2					1	1			5	2	9												
Thyroid	4	16	20			3	6	5	5	1		6																					
Toxic or Uremic	1	2	3		1	1	1																										
Emphysema	2	4	6				1		3	1	1																						
Congenital	4	3	7	2	1	2	2															INTERV. SEPTAL DEFECT 3				PATENT DUCTUS 2			ARTERIOSCLER.				
Subacute Bacterial Endocarditis	0	1	1	1																													
Diphtheritic	0	2	2		1	1																											
Pernicious Anemia	1	1	2						1	1		1																					
Sinus Arrhythmia	0	1	1	1																				1									
Effort Syndrome	26	34	60		8	19	13	13	5	2																							
Effort Syndrome + Extrasystoles	0	1	1					1																									
Irritable Heart & Extrasystoles	8	7	15		1	3	3	1	4		1																						
Irritable Heart & Paroxysmal Tachycardia	0	15	15		1	1	3	3	6	1							15																
Functional Systolic Murmurs	13	17	30	8	2	8	5	4	2	1																							
UnKnown	25	9	34			2	8	10	9	4	1	11							1	1	1			2		1							
Total in Numbers	427	573	1000	47	81	85	109	121	190	211	154	118	11	0	6	2	0	22	22	9	141	23	40	6	4	90	52	82	14	2	1	8	8	1	8
Total in Percentage	42.7	57.3	100	4.7	8.1	8.5	10.9	12.1	19.0	21.1	15.4	11.8	1.1	0.0	0.6	0.2	0.0	2.2	2.2	0.9	14.1	2.3	4	0.6	0.4	9	5.2	8.2	1.4	0.2	0.1	0.8	0.8	0.1	0.8

cent may be considered the community incidence of rheumatic heart disease among cardiac patients, comprising all the diseased patients with rheumatic etiology as a factor.

Almost twice as many cases of this group were seen in the hospital series as in general practice, and almost three times as many as in the consultation series. Of the complications subacute bacterial endocarditis comes first with 39 cases, effort syndrome, second, with 37, and arteriosclerotic (15) and hypertensive (16) heart disease each with less than half of either of the first two named. Of these patients 54.9 per cent were female and 45.1 per cent male, showing no notable variation.

This type of heart disease was commonest in the second decade (31.3 per cent), 43.4 per cent occurring in young individuals under twenty years of age. In the third and fourth decades 31.4 per cent more occurred, or 74.4 per cent under forty years of age. Twelve and nine-tenths per cent of the cases were present in the fifth decade, but it was rare after fifty years of age (12.3 per cent); one case was recorded above seventy years.

Mitral regurgitation and stenosis was the commonest valve diagnosis of the series, being present in 28.3 per cent of the cases showing valvular disease. Mitral stenosis alone (17.4 per cent) and mitral regurgitation (16.0 per cent) were next in order of frequency. It is interesting to note that aortic regurgitation alone occurred 33 times (3.5 per cent). Of the 933 cases in which it was possible to determine the organic valvular defects, they were distributed as follows:*

TABLE IV

Complete Group. 3000 Cardiac Cases.

Type	Sex		Total	Ages								Auricular Fibrillation		Block A-V		Block	Auricular Flutter	Alternation	Paroxysmal Tachycardia	Angina Pectoris	Coronary Occlusion	Pericarditis	Valve Lesions														
	Male	Female		0-10	10-20	20-30	30-40	40-50	50-60	60-70	70+	Permanent	Paroxysmal	Dig.	Org.	Intrav.							A.R.	A.S.	AR+AS	MR	MS	MR+MS	AR+MR	AR+MS	AR+AS+MR	AR+AS+MS	AR+MR+MS	AR+AS+MR+MS	AS+MR+MS	AS+MR	AS+MS
Rheumatic	363	473	836	114	278	116	134	105	63	25	1	142	16	4	16	3	2		27	7	1	61	28	5	6	134	144	238	29	60	3	7	118	36	4	2	9
Rheumatic + Arteriosclerotic	12	3	15						6	5	4	3	1		1	2				4			1	3				1		1			2	2		1	
Rheumatic + Hypertensive	8	8	16				6	2	5	3		2	2									1	1		2	2	2	5	1	1			1		1		
Rheumatic + Luetic	2	0	2					2								1											1			1							
Rheumatic + Thyroid	1	0	1				1																					1									
Rheumatic + Congenital	3	1	4		4																					1		2					1				
Rheumatic + Emphysema	0	1	1						1																												
Rheumatic + Subacute Bact. Endocarditis	22	17	39	2	15	11	8	2	1						1					1		4				4	2	13	3	3			9	1		1	
Rheumatic + Hypertensive + Luetic	0	1	1					1																													
Rheumatic + Arteriosclerotic + Hypertensive	2	1	3						1	2		1							1	1							1						1				
Rheumatic + Hypertensive + Toxic	0	1	1						1																		1										
Rheumatic + Effort Syndrome	18	19	37		2	8	16	11							1				4				3	1		8	11	4	3	6			1		1		
Arteriosclerotic	268	149	417			•	1	16	94	189	117	52	22	3	19	24		5	7	118	48		10	2		28			3								
Hypertensive	81	141	222		1	5	16	59	78	52	11	11	4			1		5	5	19	2		3			1											
Arteriosclerotic + Hypertensive	209	187	396		1			35	89	167	104	55	8		13	16	1	22	3	94	13		24			13											
Hypertensive + Uremic (Toxic)	8	5	13			2	2	3	5	0	1	2					1	1		1																	
Hypertensive + Thyroid	4	17	21		2	2	5	3	7	1	1	5							2																		
Hypertensive + Luetic	6	4	10					2	6	2		2								1			9														
Hypertensive + Emphysema	0	1	1					1																													
Hypertensive + Arteriosclerotic + Luetic	4	0	4						1	3						1		2		1			3						1								
Hypertensive + Arteriosclerotic + Uremic	3	1	4						1	1	2							1																			
Hypertensive + Arteriosclerotic + Effort Syndrome	0	1	1						1																												
Hypertensive + Effort Syndrome	3	12	15				1	5	8	1																											
Arteriosclerotic + Luetic	4	0	4							3	1	1			1	1				3			4														
Arteriosclerotic + Emphysema	7	1	8					1		4	3				1					2																	
Arteriosclerotic + Uremic	2	2	4							3	1							2																			
Arteriosclerotic + Effort Syndrome	3	5	8						3	4	1		1																								
Angina Pectoris	49	28	77			1	2	15	38	17	4									77	1																
Luetic (u ē Aneurysm)	62	12	74	1		4	11	20	26	12				1	1	3		2		23	4		51														
Thyroid	9	37	46		1	9	14	15	6	1		9	5		1				1																		
Myxoedema	0	1	1					1																													
Emphysema	3	8	11				1	1	3	4	2																										
Congenital	17	15	32	10	11	6	4	1							1																						
Congenital + Effort Syndrome	1	0	1			1																															
Uremic or Toxic	1	2	3		1	1	1																														
Subacute Bacterial Endocarditis	4	2	6	1			3	2															2						2				1				
Diphtheritic	0	2	2		1	1																															
Pernicious Anemia	1	1	2						1	1		1																									
Effort Syndrome	126	176	302	1	25	78	83	77	30	8				1					1																		
Effort Syndrome + Premature Beats	0	1	1					1																													
Irritable Heart ē Premature Beats	44	37	81	1	3	9	17	24	15	10	2		1																								
Irritable Heart ē Paroxysmal Tachycardia	28	50	78		8	9	18	18	21	4									78																		
Sinus Arrhythmia	1	2	3	2		1																				1											
Functional Systolic Murmurs	59	55	114	37	39	16	9	8	4	1																											
Unknown	57	25	82	2	1	8	14	19	27	10	1	23	7		3		2			1	2	1	2		1	5		1	1								
Total in Numbers	1495	1505	3000	171	393	288	367	450	542	533	256	309	67	9	59	52	6	46	129	353	71	67	141	11	9	197	162	260	43	72	3	7	134	39	6	4	9
Total in Percentage	49.8	50.2	100	5.7	13.1	9.6	12.2	15	18.1	17.8	8.5	10.3	2.2	.3	1.9	1.7	.2	1.3	4.3	11.8	2.4	2.2	4.7	.4	.3	6.6	5.4	8.3	1.4	2.4	.1	.2	4.5	1.3	.2	.1	.3

MCM.

1. Mitral regurgitation and stenosis, 264 (28.3 per cent).
2. Mitral stenosis, 162 (17.4 per cent).
3. Mitral regurgitation, 149 (16.0 per cent).
4. Aortic regurgitation, mitral regurgitation and stenosis, 133 (14.3 per cent).

*Abbreviations in tables for valve lesions are as follows: M.R.=Mitral regurgitation, M.S.=Mitral stenosis, A.R.=Aortic regurgitation, A.S.=Aortic stenosis.

5. Aortic regurgitation and mitral stenosis, 72 (7.8 per cent).
6. Aortic regurgitation and stenosis, mitral regurgitation and stenosis, 39 (4.2 per cent).
7. Aortic regurgitation and mitral regurgitation, 36 (3.8 per cent).
8. Aortic regurgitation, 33 (3.5 per cent).
9. Aortic stenosis and mitral stenosis, 9 (0.9 per cent).
10. Aortic stenosis, 8 (0.9 per cent).
11. Aortic regurgitation and stenosis, 8 (0.9 per cent).
12. Aortic regurgitation and stenosis, mitral stenosis, 7 (0.7 per cent).
13. Aortic stenosis, mitral regurgitation and stenosis, 6 (0.6 per cent).
14. Aortic stenosis and mitral regurgitation, 4 (0.4 per cent).
15. Aortic regurgitation and stenosis, mitral regurgitation, 3 (0.3 per cent).

Almost one-fifth (167) of the rheumatic patients showed auricular fibrillation (17.5 per cent), of which number 148 cases (15.5 per cent) were permanent, and 19 (2 per cent) exhibited the paroxysmal form of the irregularity. Two patients had auricular flutter, and 32 (3.3 per cent) paroxysmal tachycardia. Nineteen (2 per cent) showed partial auriculoventricular block apparently as a result of the disease, and four probably as a result of digitalis medication. Six cases of intraventricular block were noted.

Pericarditis was found in 66 (6.9 per cent) patients. Angina pectoris was present in 13 (1.4 per cent).

Arteriosclerotic and Hypertensive.—The greatest difficulty in the classification of the cases etiologically has been in separating the hypertensive from the arteriosclerotic group, and in diagnosing the combination. We realize the vast importance of hypertension as a cause of cardiac enlargement; yet such enlargement may occur at times without hypertension, as is noted, for instance, in cases of coro-

TABLE V

	CONSULTATION	HOSPITAL	GENERAL PRACTICE	TOTAL
	1000 I	1000 II	1000 III	3000 IV
Sex—Male	56.2	50.6	42.7	49.8
Female	43.8	49.4	57.3	50.2
Age 0–20	8.0	35.6	12.8	18.8
20–40	25.1	21.0	19.4	21.8
40–60	39.4	28.7	31.1	33.1
60–	27.5	14.7	36.7	26.3
Organic Cases	60.3	87.2	83.9	77.1
Functional Cases	33.7	12.0	12.2	19.3
Angina Pectoris (without other diagnoses)	4.2	0.7	2.8	2.6
Auricular Fibrillation (without other diagnoses)	1.8	0.1	1.1	1.0

nary thrombosis. There are certain cases belonging to each group separately, and a vast number in which the two factors must be considered together. These figures are, therefore, open to question, but they have been derived by careful analysis of each case, taking into consideration history or present evidence of hypertension, and such indications of coronary sclerosis as are obtained in older people by finding intraventricular block by electrocardiogram, tortuosity of the aorta by x-ray, beginning heart failure in old age without cardiac enlargement or hypertension, and by clear evidence of acute coronary thrombosis. With allowances for some error, we have considered that all cases of the organic group showing either arteriosclerosis or hypertension or both constitute 50.3 per cent (1,164 cases). This would constitute 38.8 per cent of the total series (organic and functional). The group considered as an *entity* is the largest by far of any in the series.

The arteriosclerotic group alone comprised the greatest number of cases 417 (18 per cent of organic group; 13.9 per cent of total series) of this division. To this must be added the large group having both arteriosclerotic and hypertensive factors, 396 (17.1 per cent of organic group; 13.2 per cent of total series), and 51 (2.2 per cent of organic group; 1.7 per cent of total series) patients having other complications. Thus, the total group exhibiting arterioscloresis as a factor is 864 (37.3 per cent of organic group; 28.8 per cent of total series). The community incidence of arteriosclerotic heart disease may be considered as 37.3 per cent of individuals with cardiac disease. There is a predominance of the males (59.6 per cent) over females (40.4 per cent). The age incidence is as expected. Only two cases occurred prior to forty years (0.2 per cent) of age. In the fifth decade there were 52 cases (6.1 per cent). The remaining 810 cases (93.7 per cent) occurred above the fifth decade, being fairly evenly distributed. The seventh decade included 381 cases (44.1 per cent).

The chief complications along with arteriosclerotic heart disease, omitting the hypertensive type, were rheumatic heart disease (15), emphysema (8), effort syndrome (8), luetic heart disease (4), uremic or toxic heart disease (4), hypertensive and luetic heart disease (4), rheumatic and hypertensive heart disease (3), and effort syndrome and hypertensive heart disease (1).

Auricular fibrillation was present in 144 patients (16.7 per cent), of which number 32 cases were paroxysmal in nature; auriculoventricular block occurred in 34 patients (3.9 per cent); intraventricular block in 45 patients (5.2 per cent); paroxysmal tachycardia in 11 (1.3 per cent); and auricular flutter in one.

Alternation of the pulse was noted in 32 patients (3.7 per cent), all but 7 of the patients having hypertension also. Angina pectoris was extremely common, 223 cases (25.8 per cent); occurring 118 times

in patients with arteriosclerotic heart disease alone. Coronary occlusion was also common, 61 cases (7.1 per cent); 48 being in patients with "pure arteriosclerosis."

The group exhibiting *hypertension* as the only etiological factor was about half the size of the pure arteriosclerotic group. There were 222 such cases (9.6 per cent of organic cases; 7.4 per cent of total series). To obtain the true incidence of hypertensive heart disease, we must add the large hypertensive and arteriosclerotic group of 396 cases (17.1 per cent of organic cases; 13.2 per cent of total series), and the other complications, 90 cases (3.9 per cent of organic cases; 3 per cent of total series). Thus, the total hypertensive series consists of 708 cases (30.6 per cent of organic cases; 23.6 per cent of total series). The community incidence of hypertensive heart disease may be considered as 30.6 per cent of cardiac patients.

Females (54.7 per cent) were preponderant over males (45.3 per cent). The sixth (28.7 per cent, or 203 cases) and seventh decades (32.8 per cent, or 232 cases) contained 61.5 per cent of the cases. There were four patients under twenty years (0.5 per cent; two of the patients had hyperthyroidism); 9 (1.3 per cent) in the third decade; 14 (4.2 per cent) in the fourth decade; 111 (15.7 per cent) in the fifth decade; and 119 (16.8 per cent) above seventy years.

Ninety-two patients (13 per cent) had auricular fibrillation, fourteen of the cases being paroxysmal. Auriculoventricular block was found in 13 patients (1.8 per cent), and intraventricular block occurred 18 times (2.5 per cent). Paroxysmal tachycardia was seen in 11 patients (1.5 per cent), and auricular flutter in 2 (0.3 per cent). Alternation of the pulse was relatively common, 31 (4.4 per cent). Angina pectoris was present in 117 patients (16.5 per cent), but all but 21 of the 117 patients exhibited arteriosclerosis as an additional etiological factor. Coronary occlusion occurred in 15 patients (2.1 per cent).

Luetic.—The luetic heart disease group (aortitis with or without cardiac involvement) does not occupy as prominent a part in the series as had been expected. Altogether there were 95 cases (4.1 per cent of organic cases; 3.2 per cent of total series), giving a community incidence of 4.1 per cent of cardiac patients. Of these 95 patients, 21 had additional etiological factors: hypertensive heart disease in 10; arteriosclerotic heart disease in 4; arteriosclerotic and hypertensive heart disease in 4; rheumatic heart disease in 2; and rheumatic and hypertensive heart disease in 1. There were four males (82.1 per cent) to every female (17.9 per cent), showing a very marked predominance as to sex. The vast majority of cases occurred in the fifth (26.2 per cent), sixth (34.7 per cent), and seventh (21.5 per cent) decades, 82.4

per cent of the cases falling in these age groups. The first decade included 1 case (1 per cent), the third, 4 (4.1 per cent), and the eighth, 1 (1 per cent).

Aortic regurgitation was present in 69 of the 95 cases. One of the patients was diagnosed as having mitral regurgitation also, and one, mitral stenosis. Auricular fibrillation occurred in 3, auriculoventricular block in 2, intraventricular block in 5, and alternation of the pulse in 4. Angina pectoris was common, occurring in 27 patients (28.4 per cent). Coronary occlusion was found 4 times (4.1 per cent).

Thyroid.—Hyperthyroidism was an etiological factor in 68 cases in the series (2.9 per cent of organic group; 2.3 per cent of total series). Almost one-third of these patients (21) had, in addition, a hypertensive factor. In one patient there also was a rheumatic etiological basis. Females (79.4 per cent) predominated over males (20.6 per cent) about four to one. A fairly even distribution as to age occurred in the third (16.2 per cent), fourth (29.4 per cent), fifth (26.5 per cent), and sixth (19.1 per cent) decades, the fourth and fifth decades including over half the group (55.9 per cent). Auricular fibrillation was present in about one-fifth of the cases (14 or 20.6 per cent); one-third of these (5) being paroxysmal. One patient exhibited auriculoventricular block, and 3 (4.4 per cent), auricular paroxysmal tachycardia.

Myxedema as the cause of organic heart disease has been the subject of much discussion. One such case occurred in the series, falling in the category described by Fahr[8] and by Means, White and Krantz.[9] The case would represent 0.04 per cent of the organic group and 0.03 per cent of the total series.

Emphysema.—A relatively unimportant factor etiologically is emphysema. There were 21 cases (0.9 per cent of the organic group; 0.7 per cent of total series) in which it seemed to be a factor. In half of these cases (10) it aggravated another etiological factor (arteriosclerotic, 8; hypertensive, 1; and rheumatic, 1). There was no sex predominance (10 males, 11 females). It is a disease of the later decades of life. One case occurred in the fourth decade, 3 in the fifth (14 per cent), and 17 (81 per cent) in the sixth (19 per cent), seventh (38 per cent), and eighth (24 per cent) decades.

Congenital.—Thirty-seven cases (1.6 per cent of organic group; 1.2 per cent of total series) were included in the series, showing that such cases are not nearly so rare as might be expected. Fifty-six and seven-tenths per cent were males, and 43.3 per cent, females. Twenty-seven per cent occurred in the first, 40.6 per cent in the second, 18.9 per cent in the third, 10.8 per cent in the fourth, and 2.7 per cent in the fifth decades. In 35 cases a tentative diagnosis of the defect was made:

intraventricular septal defects 12, patent ductus arteriosus 11, pulmonary stenosis 8, and combinations of the above 4. It is interesting to note that in four patients there was also a rheumatic factor.

Uremic or Toxic.—Such a condition as an etiological factor per se is rare. However, as an added factor in the case of a previously damaged cardiovascular system, it is a more significant finding. Twenty-five patients (1.1 per cent of organic group; 0.8 per cent of total series) had this factor, only three exhibiting it alone. It was seen commonest along with hypertension, three-fourths of the cases (18) showing also hypertensive heart disease. It was present in 4 patients with arteriosclerotic heart disease, in 4 with hypertension and arteriosclerosis, and in 1 with rheumatic type of cardiac disease. Fifty-six per cent of the patients were males; 44 per cent were females. After the second decade, there is little difference in age incidence. Two patients showed auricular fibrillation, 1 auricular flutter, 4 alternation of the pulse, and 1 had angina pectoris.

Subacute Bacterial Endocarditis.—With a rheumatic background this is a fairly common condition, having occurred in 39 patients of the rheumatic group. Six cases are also recorded in which there was no previous demonstrable lesion. Forty-five patients (1.9 per cent of organic group; 1.5 per cent of total series) in all are included in the series. One and nine-tenths per cent would be considered its community incidence among cardiac patients. Of the 956 rheumatic heart disease cases, 4.1 per cent had also subacute bacterial endocarditis, showing that it is the terminal condition in a goodly number of this series. Of these cases 57.7 per cent occurred in males, and 42.3 per cent in females. Eighty-two and three-tenths per cent of the cases occurred in the second (33.3 per cent), third (24.5 per cent), and fourth (24.5 per cent) decades. In the first (6.6 per cent), fifth (8.9 per cent), and sixth (2.2 per cent) decades, there were few cases.

Diphtheria.—Two cases (0.08 per cent of organic group; 0.07 per cent of total series) are recorded in the general practice group of organic heart disease resulting from diphtheria during the acute infection.

Pernicious Anemia.—In two cases (0.08 per cent of organic group; 0.07 per cent of total series) pernicious anemia is given as the etiological basis of organic heart disease. Both cases occurred in the general practice group.

FUNCTIONAL HEART DISORDERS

Effort Syndrome.—Easily the commonest functional disorder encountered fell under this group. There were 302 cases of effort syndrome alone, and one case accompanied by another functional disorder, premature beats, making the total of purely functional effort

syndrome cases, 303 (52.3 per cent of functional group). In addition to this there were 62 cases in which effort syndrome was present along with organic heart disease, making the total 365 cases (12.2 per cent of total series). Two and seven-tenths per cent of all the organic cases (2,314) had the additional entity of effort syndrome. Over one-half (37 of 62) of the patients who showed both effort syndrome and organic heart changes had rheumatic heart disease, and about one-fourth (15 of 62) had hypertensive heart disease. Over three-fourths (77 per cent) of the total cases occurred in the third (23.9 per cent), fourth (27.4 per cent), and fifth (25.7 per cent) decades. It was rare in the first (0.3 per cent) and eighth 0.3 per cent decades. In the second (7.4 per cent), sixth (11.5 per cent), and seventh (3.5 per cent), it was moderately common. Females (58.6 per cent) were slightly predominant over males (41.4 per cent).

Irritable Heart With Premature Beats.—Almost one-sixth (82 or 14.2 per cent) of the functional cases were those exhibiting irritable hearts with premature beats. The number of cases of organic heart disease at one time or another showing premature beats was not recorded. Premature beats alone (one case also had effort syndrome) occurred in 2.7 per cent of the total series. There was no especial sex predominance (males 53.7 per cent; females 46.3 per cent). The fourth, fifth, and sixth decades include over three-fifths (69.5 per cent) of the cases. The percentages for each decade are as follows: first, 1.2 per cent; second, 3.7 per cent; third, 11.0 per cent; fourth, 20.7 per cent; fifth, 30.5 per cent; sixth, 18.3 per cent; seventh, 12.2 per cent; and eighth, 2.4 per cent.

Irritable Heart With Paroxysmal Tachycardia.—Hardly less common than premature beats as a functional disorder in patients without organic heart disease was paroxysmal tachycardia. It was present in 78 patients as the only diagnosis and along with effort syndrome in one, a total of 79 (13.6 per cent of functional series) in purely functional cases. The condition was more common in females (64.1 per cent) than in males (35.9 per cent). The age incidence by decades was as follows: second, 10.3 per cent; third, 11.5 per cent; fourth, 23.1 per cent; fifth, 23.1 per cent; sixth, 26.9 per cent; and seventh, 5.1 per cent. In 50 cases of organic heart disease this disorder was also noted, making the total 129 cases (4.3 per cent of total series). It was most commonly seen along with rheumatic heart heart disease (32 cases, 64.0 per cent). Thus 3.3 per cent of the rheumatic group showed the disorder. In 18 cases (30.0 per cent), the concurrent type of heart disease was arteriosclerotic or hypertensive, separately, in combination, or with another additional organic factor.

Sinus Arrhythmia.—In only 3 cases (0.5 per cent of functional; 0.1 per cent of total series) was sinus arrhythmia sufficiently marked to

cause its special notation. Two of these cases fell in Group I, having been referred to a cardiac consultant because of questionable cardiac disease.

Functional Systolic Murmurs.—A relatively common group in the series was composed of those cases showing functional systolic murmurs. These murmurs may be described as those apical or basal systolic murmurs varying during respiration or on changes in posture and rate, not masking or altering the first sound, and not associated with cardiac enlargement or other evidence of organic cardiac disease. There were 114 such cases (19.7 per cent of functional group; 3.8 per cent of total series). Well over half the cases (62) were present in Group II, being children or young adults referred to the cardiac clinics for questionable heart disease. There was no sex variation (male 51.8 per cent; female 48.2 per cent). Two-thirds of the patients (66.7 per cent) were under twenty years of age. By decades the percentages were as follows: first, 32.5 per cent; second, 34.2 per cent; third, 14.0 per cent; fourth, 7.9 per cent; fifth, 7.0 per cent; sixth, 3.5 per cent; and seventh, 0.9 per cent.

UNKNOWN

Organic heart disease of unknown etiology was present in 52 patients (2.2 per cent of organic group; 1.7 per cent of total series). In addition in the tables, 30 cases of auricular fibrillation without other diagnoses are recorded, and they will be taken up under the appropriate heading. Of the unknown etiology cases 10 (19.2 per cent) had various valvular diagnoses; 3 (5.8 per cent) had auriculoventricular block; 2 (3.8 per cent) had auricular flutter; 2 (3.8 per cent) had coronary occlusion; 1 (1.9 per cent) had angina pectoris, and 1 (1.9 per cent) had pericarditis.

ANGINA PECTORIS

There were 77 patients who had angina pectoris without any demonstrable etiological basis. The condition was much more common along with organic heart disease, being noted 276 times in this connection. The total incidence of angina pectoris in the series is 353 (11.8 per cent of total series). One-third of all the cases (118 or 33.4 per cent) occurred with arteriosclerotic heart disease, and the various etiological combinations in which arteriosclerosis figured brings the number to 223 (63.2 per cent), three-fifths of all the cases exhibiting angina pectoris. Hypertension (and its complications) was next in order with 117 cases (33.1 per cent), but about four-fifths of these cases had an arteriosclerotic factor in combination with the hypertension. Only 19 cases (5.3 per cent) of apparent hypertensive heart disease alone had angina pectoris. Angina pectoris occurred in

28 patients (7.9 per cent) having luetic heart disease, 23 of the patients (6.5 per cent) having this alone as an etiological factor. In rheumatic heart disease alone angina pectoris is not so common, occurring in 7 patients (2.0 per cent), while with its complications (chiefly arteriosclerotic) it is not quite so rare, 7 cases (3.9 per cent).

CORONARY OCCLUSION

In 71 patients (3.1 per cent of organic; 2.4 per cent of total series), coronary occlusion occurred. At least fifty-two of the patients (87.3 per cent) had an arteriosclerotic basis (13 of this group also had hypertension). In 4 cases (5.7 per cent) of luetic heart disease it was found, and in one case (1.4 per cent) of rheumatic heart disease. In two cases (2.8 per cent) the etiology was unknown.

CARDIAC IRREGULARITIES

Auricular Fibrillation.—Since the status of auricular fibrillation as a functional or organic condition is interpreted variously by students of heart disease, 30 cases of this irregularity, showing no other cardiac abnormality and charted in the tables under the heading unknown, were considered as a separate group. When organic heart disease cases showing the irregularity are considered, the frequency increases tremendously. There were 376 cases in all (12.5 per cent of total series), 309 (82.2 per cent) being permanent and 67 (17.8 per cent) paroxysmal in type. Of the 346 patients having the irregularity along with organic heart disease, the rheumatic group is easily the most important, almost half the cases (158 or 45.6 per cent) occurring in this type of cardiac disease. It was present in 167 cases (48.3 per cent) of the combined rheumatic group. It was common along with arteriosclerosis (74 cases or 21.4 per cent), reaching a total of 144 cases (41.7 per cent), when we consider also the complications of arteriosclerosis. It was less common with hypertensive hearts (15 cases or 4.3 per cent), but the combined group showed the irregularity in 92 cases (26.6 per cent). In 14 patients (4.0 per cent) thyroid heart disease was present, and adding the thyroid cases with hypertension it occurred 19 times (5.4 per cent).

Heart-Block.—A. Auriculoventricular: This study does not give a correct idea of the prevalence of this condition since electrocardiographic studies were not made of the entire group. The great majority of Groups I and II, especially those patients likely to show this abnormality, were electrocardiographed, but very few of Group III were so studied. The condition was found in 59 cases (2.6 per cent of organic group; 1.9 per cent of total cases). In 35 cases (59.3 per cent) there was an arteriosclerotic basis; 19 (32.2 per cent) rheu-

matic; 3 (5.1 per cent) unknown; 1 (1.7 per cent) congenital; 1 (1.7 per cent) thyroid, and 1 (1.7 per cent) luetic. In addition to the above cases there were 9 cases exhibiting this phenomenon as a result of digitalis medication.

B. Intraventricular: This group is inaccurate for the reason cited in the case of auriculoventricular block above. Fifty-two cases (2.2 per cent of organic group; 1.7 per cent of total series) were found. Four-fifths of the cases (44 or 84.6 per cent) had an arteriosclerotic basis (including arteriosclerotic cases and those with complications). In 3 cases (5.8 per cent) of uncomplicated rheumatic heart disease, and in 3 cases (5.8 per cent) of uncomplicated luetic heart disease the abnormality was found. The condition was present only once (1.9 per cent) in uncomplicated hypertensive heart disease.

Auricular Flutter: This rare condition was noted only 6 times (0.3 per cent of organic cases; 0.2 per cent of total series) in the entire series. Two of the patients had rheumatic heart disease, one had arteriosclerotic and hypertensive heart disease, one had hypertensive and uremic or toxic heart disease, and two had cardiac disease of unknown etiology.

Alternation of the Pulse: In 40 cases (2.2 per cent of organic group; 1.7 per cent of total series) alternation of the pulse was noted. Thirty-eight (95 per cent) of the patients had arteriosclerosis, hypertension, or both. There was little difference between the groups. It was noted twice (5.0 per cent) in luetic patients, and in four patients (10.0 per cent) there was a toxic or uremic factor along with hypertensive and arteriosclerotic heart disease.

SUMMARY AND CONCLUSIONS

1. An analysis of 3,000 patients in New England seeking medical advice for cardiac symptoms or signs in the past few years has shown that four-fifths (80.7 per cent) had organic heart disease, if one includes cases with uncomplicated angina pectoris (2.6 per cent) and uncomplicated auricular fibrillation (1.0 per cent). One-fifth (19.3 per cent) had relatively unimportant functional circulatory abnormalities. In the text, uncomplicated angina pectoris and uncomplicated auricular fibrillation are considered separately.

2. In the group of 2,421 cases of organic heart disease, including the patients with uncomplicated angina pectoris (77) and uncomplicated auricular fibrillation (30), the etiological factors apparently present, both alone and complicated, were found to be as follows in the order of frequency:

(a) "Rheumatic," 956 cases or 39.5 per cent.

(b) Arteriosclerotic, 864 cases or 35.7 per cent.*

(c) Hypertensive, 708 cases or 29.2 per cent.*

(Hypertensive or arteriosclerotic or both, 1,164 cases or 48.1 per cent.)*

(d) Angina pectoris, 353 cases or 14.6 per cent.

(e) Syphilitic, 95 cases or 3.9 per cent.

(f) Coronary occlusion, 71 cases or 2.9+ per cent.

(g) Hyperthyroid, 68 cases or 2.9 per cent.

(h) Subacute bacterial endocarditis, 45 cases or 1.9 per cent.

(i) Congenital, 37 cases or 1.5 per cent.

(j) Miscellaneous, 51 cases or 2.1 per cent.

(k) Unknown (including the 30 cases of uncomplicated auricular fibrillation) 82 cases or 3.4 per cent.

Frequently two and sometimes three of these etiological factors appeared to be combined in the same case.

3. In the group of 579 cases with relatively unimportant functional circulatory abnormalities and without evidence of heart disease the various disturbances were represented, alone or in combination, as follows:

(a) Effort syndrome, 303 cases or 52.3 per cent.

(b) Functional systolic murmurs, 114 cases or 19.7 per cent.

(c) Premature contractions, 82 cases or 14.2 per cent.

(d) Paroyxsmal tachycardia, 79 cases or 13.6 per cent.

(e) Sinus arrhythmia (marked), 3 cases or 0.5 per cent.

These functional disturbances were often associated also with organic heart disease.

4. Auricular fibrillation occurred altogether in 376 cases of the 3,000, thirty times apparently uncomplicated. In 67 patients it was paroxysmal and in 309 permanent in type. Auricular flutter was found in only 6 patients.

5. Auriculoventricular heart-block was found in 59 patients, undoubtedly an underestimate. Intraventricular block was found in 52 patients. Since electrocardiograms are necessary to discover this last mentioned condition and since less than half of the cases were electrocardiographed it should be considered relatively common.

6. Alternation of the pulse was noted in only 40 patients, all with serious organic heart disease. Undoubtedly it was often overlooked.

*It is quite possible that the arteriosclerotic factor may be somewhat overestimated in this study and that the hypertensive element may be underestimated. That is, past hypertension of which there is no history, with normal blood pressure at the time of examination, may of course have been overlooked. Finally, in either case, especially in the arteriosclerotic group, some other unknown factor or factors may have existed. However, as stated in the text, as careful an attempt as possible in the absence of necropsy confirmation, has been made to determine the presence or absence of the arteriosclerotic factor.

7. One of the most important objects and results of this study has been the comparison of the three different groups; namely, that of 1,000 cases from private consultation practice, that of 1,000 cases from the hospital and dispensary, and finally that of 1,000 cases from general practice. Such a comparison has been neglected in the past; too often hospital statistics, relatively easily obtained, have been more or less unconsciously considered to be community statistics. Eventually, when our hospitals do represent quite accurately all classes of the community in proportion, then hospital statistics in themselves will suffice. In the past it has been true that statistical studies from public hospitals have been generally more reliable than those from private practice, but that need not be so, and the reverse is sometimes true.

In the present study patients with organic heart disease were more common in the hospital group than in private practice; patients with relatively unimportant functional cardiovascular disturbances much more frequently consulted the specialist, perhaps because they were better able to do so. Thus, uncomplicated effort syndrome was found in 207 cases in private consultation, in 60 cases in general practice, and in 35 cases in the hospital group.

Also angina pectoris was found much more frequently in private practice, being recorded in 141 cases of the general practice series, in 129 cases of the consultation group, and in 83 cases in the hospital.

On the other hand rheumatic heart disease was much more common in the hospital and dispensary group than in private practice, being found in about half of the former (474 cases) as contrasted with 282 cases from general practice and 200 cases seen in private consultation. This undoubtedly accounts for the greater number of children seen in the hospital and dispensary group. Rheumatic heart disease tends to be a disease of relative poverty.

The syphilitic factor, as expected, was present more often in the hospital class, but even there it was surprisingly infrequent. It occurred in 43 cases of the hospital group, in 28 cases in general practice and in 23 cases seen in private consultation. We are quite sure that not many cases were overlooked.

Arteriosclerosis as a factor was more frequent in private than in hospital practice. It appeared to be present in 258 patients in general practice, in 276 in consulting practice, and in 230 of the hospital group (in spite of the higher percentage of organic heart disease in this last group).

Hypertension as a factor was frequent in all groups, being found in 304 cases in general practice, in 231 cases in the hospital and dispensary, and in 173 cases in consulting practice.

8. Studies similar to this, preferably with more post-mortem confirmation, in other parts of the world should reveal many facts of

interest concerning the relative incidence of types of heart disease in different climates, countries, and classes of society. Comparison of the hospital incidence of heart disease elsewhere may be made with the hospital group of the present series; in this general hospital (Massachusetts General Hospital) of 425 beds, 500 consecutive cardiac ward cases were collected out of a total of 2,766 medical and 8,226 total admissions in fourteen months. To this hospital came patients from all over New England.

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SUPRAVENTRICULAR PAROXYSMAL TACHYCARDIA WITH A VARYING SITE OF STIMULUS ORIGIN*

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PAROXYSMAL tachycardia has been known for many years; and since the introduction of the electrocardiograph it has become possible to differentiate several varieties depending on the site from which the stimulus arises which controls the heart. The stimulus may arise in the auricle, in the auriculoventricular node, or in the ventricle. The majority of cases are of supraventricular origin, and many reports of paroxysms of this nature have been published. The literature relating to the subject has been fully reviewed recently by Lewis[6] and by Ritchie.[10] Paroxysms start and end suddenly. They may take place in patients with heart disease and also in individuals who show no cardiac abnormality in the interval between attacks. The attacks may be brought on by a variety of causes, such as gastrointestinal disorders, emotion, or exercise, but in many cases no cause can be found to which the onset of a paroxysm can be attributed. The duration of the paroxysms varies. The stimulus usually arises from a single abnormal focus, and the electrocardiographic curves show a constant form. The heart rate remains constant during a paroxysm although rare instances have been described in which this was not so. Frequently in the intervals between attacks isolated extrasystoles may be seen which, as a rule, originate from the same focus as the paroxysm.

The case which is described below is of interest because, during a paroxysm of tachycardia of supraventricular origin induced by exercise, the site from which the stimulus arose which controlled the heart appeared to change throughout the paroxysm. Many paroxysms, the duration of which varied between fifteen and forty-five minutes, have been studied in this subject. Striking changes took place in the electrocardiographic records during the course of a paroxysm, and these were constant in each instance.

CLINICAL NOTES

E. M. F., aged twenty-five years, a medical student, complained of attacks of palpitation of the heart. These commenced between the ages of ten and twelve. They were brought on by either excitement or exercise and were particularly liable to occur when a combination of these factors existed. At first they

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†Working on behalf of the Medical Research Council.

were of frequent occurrence, but of late years they have been infrequent and are now only induced by violent exercise, such as football. They are more likely to occur immediately after a meal or when indigestion is present. The attacks begin and end abruptly. Their duration varies, but, as a rule, they last from fifteen to forty-five minutes although sometimes they continue as long as the provoking factor is present. Soon after the patient began to suffer from the attacks, he discovered that they could be terminated by taking a deep breath. The ease with which this took place varied, but he noticed that in the earlier stages of a paroxysm a deep breath was usually without effect. The patient could tell the time at which he thought a deep breath would be effective in stopping a paroxysm. In the interval between attacks he felt perfectly normal, and during an attack his only symptom was palpitation. There was no dyspnea, precordial pain, or faintness. After the cessation of a long paroxysm he sometimes felt a little tired. There was

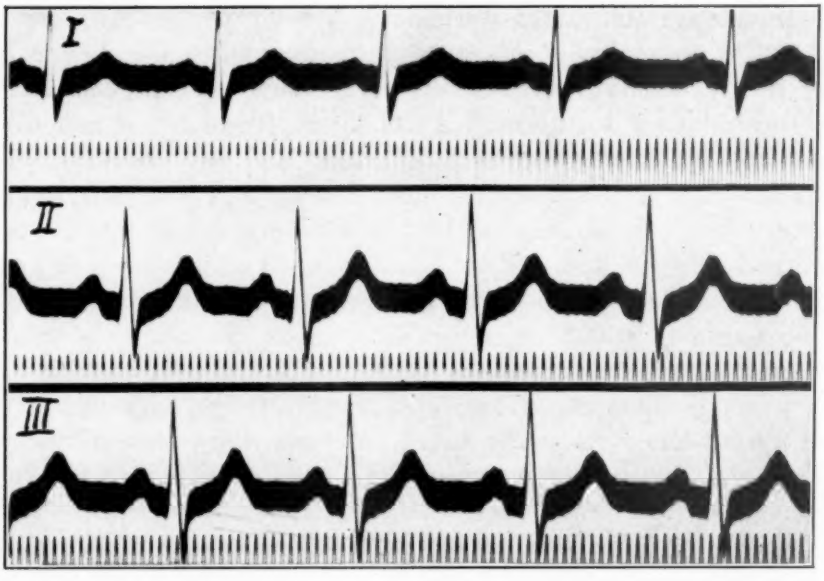


Fig. 1.—Leads I, II, III. Normal rhythm. In all records time marker is 28.57 vibrations per second. 1 cm. = 1 millivolt.

no history of rheumatic fever, scarlet fever, or repeated tonsillitis. The family history was negative. The patient started smoking at the age of twelve, and since parental permission was obtained has smoked heavily. He states that cigarette smoking has a definite influence on the frequency of the attacks.

The physical examination under normal conditions was entirely negative. During a paroxysm there was marked pulsation in the jugular veins and also over the precordium, particularly in the region of the apex. No thrill was present and the heart did not increase in size. No murmur was heard in any area.

ELECTROCARDIOGRAPHIC RECORDS

The attacks which have been investigated were induced by running up and down a long flight of stairs. The patient states that he can easily determine the moment at which a paroxysm starts, as he has a sensation which differs entirely from the tachycardia due to exercise.

On this sensation being observed electrodes were applied as quickly as possible, and electrocardiograms were taken at frequent intervals throughout the paroxysm.

When normal rhythm was present, the heart rate varied between 60 and 70 per minute, the P-R interval was 0.17 of a second, and the electrocardiogram showed no abnormality. (Fig. 1.) After the attack commenced the heart rate increased to 181 per minute, and for the first two minutes the records showed the P- and T-waves superimposed. (Fig. 2.) P can be seen definitely notching T in many of the complexes. The ventricular complexes were of the supraventricular type, and the heart beats followed each other at equal intervals. In a rec-

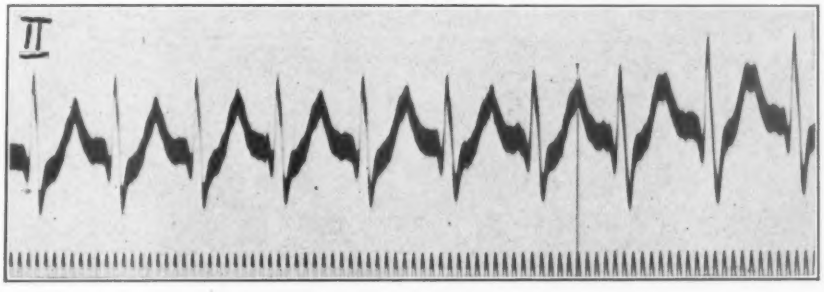


Fig. 2.—Lead II. Two minutes after the onset of the paroxysm. (For description see text.)

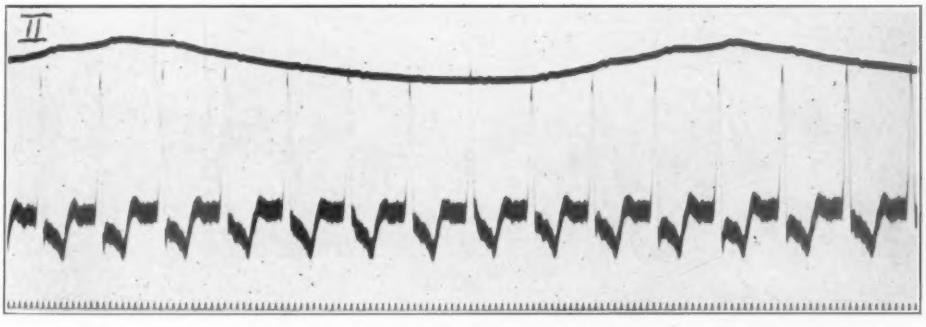
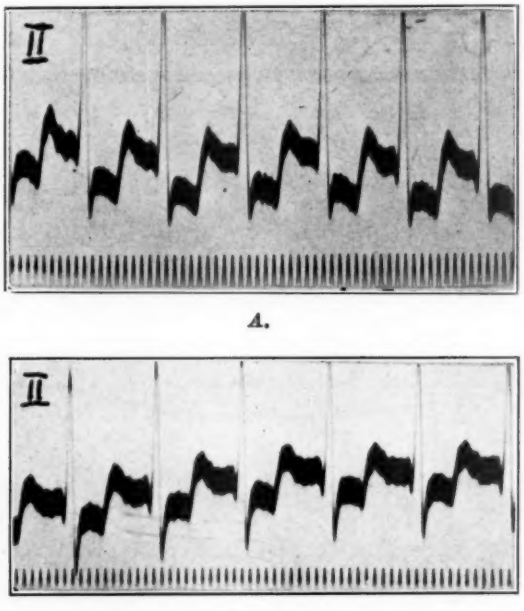


Fig. 3.—Lead II. Three minutes after the onset. Respiratory record above. Upstroke is inspiration and downstroke is expiration. (Reduced to ¾.)

ord taken three minutes after the paroxysm started the picture had completely changed. (Fig. 3.) The heart rate was 175 per minute. During expiration S was absent and R was followed by a deep negative deflection which in turn was succeeded by a slight positive wave. During inspiration, however, a small S was seen before the negative deflection took place. The question which naturally arises is whether the negative wave is an inverted P, whether it is an inverted T and the positive wave which follows is P, or whether both waves are parts of a diphasic T with P buried in the ventricular complex. It seems probable that the negative deflection was at least in part an inverted P on account of its form and also because even when no S was pres-

ent a notching was seen which strongly suggests that another wave had been superimposed on the ventricular complex. This notching is particularly well seen in the ninth beat illustrated. The positive wave, although not marked, closely resembles the finish of T in the record obtained during the cessation of a paroxysm. (Fig. 5.) During expiration at this period well-marked alternation of the ventricle was seen, but this disappeared during inspiration. Throughout the remainder of the paroxysm, less marked, but nevertheless significant, changes took place. One minute after the last record the heart rate was 176 per minute and a well-marked S was present during both phases of respiration. (Fig. 4-*A*.) The negative wave after S was much less prominent while the positive wave which followed it was



A.

B.

Fig. 4.—*A*, Lead II. Four minutes after the onset. *B*, Lead II. Sixteen minutes after the onset.

more marked. Gradually the tracing changed to that shown in the next record (Fig. 4-*B*) which was taken twelve minutes later. The rate was 163 per minute, and the complexes were of the same form except that the negative deflection was now almost absent while in later records it became completely absent. Finally the end of the paroxysm is shown. (Fig. 5.) The paroxysmal rate was 163 per minute and no negative wave followed S. This record shows clearly that the positive wave after S was T and that no sign of P was present. The end of the paroxysm was followed by a postparoxysmal pause of long duration. This pause was equal to slightly over four paroxysmal beats. On the resumption of normal rhythm there was a gradual quickening of the heart until the normal rate was attained.

Vagal Stimulation.—Pressure on the vagus in the neck and ocular compression were without effect in every instance, but these, as a rule, were applied in the early stages of the paroxysm. Undoubtedly the vagus had a definite influence as every paroxysm was stopped by a deep breath. It is interesting that in the early part of an attack this was ineffective, and that the patient was able to recognize the point at which it was likely to be effective.

Atropine.—In order to study whether vagal paralysis could induce a paroxysm $\frac{1}{50}$ of a grain of atropine sulphate was given hypodermically. The heart rate increased from 61 to 95 per minute, but no electrocardiographic change was observed except the usual shortening of the diastolic period.

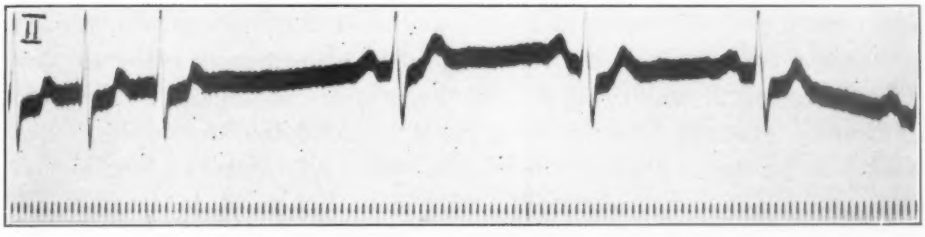


Fig. 5.—Lead II. Thirty-five minutes after the onset, as the result of a deep breath, reversion to normal rhythm took place. (Reduced to ¾.)

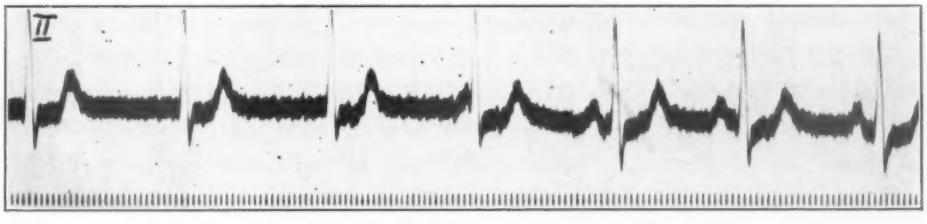


Fig. 6.—Lead II. Reversion to normal rhythm after nodal rhythm had been produced by adrenalin. (Reduced to ¾.)

Adrenalin.—An extremely interesting result followed the hypodermic injection of adrenalin, m. xv. The normal heart rate was 63 per minute. The injection was given at 4:26. At 4:28 the rate had increased to 81, but the complexes were still of normal form. A record taken at 4:30 showed an entirely different picture. The rate was now 86 and P was completely absent while T had increased greatly in height. At this time and for the next few minutes the patient said his sensations were similar to those experienced during a paroxysm only more marked. Subsequent records which were taken at frequent intervals showed a gradual slowing of the rate to 75 per minute and a lessening of the height of T but P was still absent. At 4:43 the heart reverted to normal rhythm. (Fig. 6.) The first three beats shown resemble those described above. The fourth shows a positive

P appearing close to the ventricular complex, the P-R interval being 0.09 of a second. Normal rhythm was restored in the next beat. There was no pause during the reversion to normal rhythm.

DISCUSSION

The first interesting feature of this case is the striking changes which took place in the electrocardiographic records during a paroxysm. These were due to the fact that the stimulus to contraction did not arise from a constant focus but varied its position throughout the attack. During the first two minutes of the paroxysm there seems little doubt that the impulse arose in the sino-auricular node or very close to it. The records which were obtained thereafter showed, however, that the site had changed. The interpretation of these records has been discussed above, and the conclusion was reached that P had shifted so that it now followed R. It had also become inverted, and a Vs-As interval was present. As the attack proceeded the inverted P became gradually less evident until in the later stages no P was seen. It was now probably buried in the QRS complex. During all this period the heart seemed to be responding to stimuli arising in the A-V node. In their experimental work on animals, Meek and Eyster[9] studied the electrocardiographic changes which took place when A-V rhythm was induced by various methods. They found that the relation of P to QRS was not constant. In some experiments P preceded R but the P-R interval was short, in others no P was visible, while in a further group P followed R. In certain experiments the position of P shifted during the time A-V rhythm was present. Their explanation of these changes was that the impulse may arise at different points in the A-V node. If it arises in the upper part of the node near the coronary sinus, there is a shortened As-Vs interval, if about the middle of the node, the auricle and ventricle begin to contract synchronously so that P is buried in QRS, while if in the ventricular portion of the node a Vs-As interval is present. Cases of nodal rhythm in man have been described by Mathewson[8] and Cohn[1] in which the relation of P to R varied during the time the heart was responding to the A-V node, but in both these instances the heart rate was slow, while in the present case the heart rate was considerably increased. The probable explanation of the series of changes which took place is that the attack started as a sinus tachycardia but soon the lower part of the A-V node took control. As the paroxysm continued the site of stimulus formation gradually shifted up the node towards the auricle, and finally a reversion to normal rhythm took place.

As a rule in paroxysmal tachycardia the heart rate remains constant throughout the attack. Feil and Gilder[2] found in the cases of

auricular or A-V origin which they investigated that the action of the heart was remarkably regular during a paroxysm. Cases have, however, been recorded by Iliescu and Sebastiani,[4] Strong and Levine,[13] Marvin,[7] and Gilchrist[3] in which this was not so. In the first instance the paroxysms were of auricular origin and slowing was induced by quinidine, while the other authors described ventricular paroxysms in which the changes in rate took place spontaneously. In the present case during the period when the sinus was in control, the heart rate was 181 per minute. On the shift to the A-V node taking place the rate slowed slightly to 175 per minute. During the rest of the paroxysm the rate gradually slowed to 163 per minute. These variations in rate were less than those described by the above authors but were nevertheless definite.

After the injection of adrenalin a paroxysm was not induced, but A-V rhythm became established. No shifting of the site of impulse formation took place during this period except that during the transition to normal rhythm there was one beat in which the auricle probably responded to the sinus, and the ventricle to the A-V node as was shown by the presence of a positive P with a P-R interval of 0.09 of a second. The beat before this showed A-V rhythm and that which followed showed normal rhythm. The usual action of adrenalin on the heart is an increase in rate, but the tachycardia is usually of sinus origin. There may also be a slight shortening of the As-Vs interval. Scott,[12] however, described a case of ventricular tachycardia in man in which short paroxysms could be induced by adrenalin. Kahn[5] in his experiments on animals produced complete heart-block and also ventricular extrasystoles by injection of adrenalin. The doses which he gave were very large and not comparable to those used in man. He also found changes in the T-wave. If it were negative before the injection, it became either isoelectric or even positive, while if it were positive its height was increased. In the present case a definite increase in the height of T was observed.

There are various methods by which A-V rhythm may be induced. First, by a reduction in the rate of stimulus production in the sinus so that the impulses generated in the A-V node control the heart, although the rhythmicity of the latter center is not increased. In this type the heart rate is slower than normal. Second, by an increase in the rhythmicity of the A-V node so that its rate of impulse formation exceeds that of the sinus. In this instance the rate of the heart is more rapid than normal. Third, one may have a combination of the other two factors and the heart rate is variable. Rothberger and Winterberg,[11] in their study of the influence of the vagi and sympathetic nerves on the heart, found that A-V rhythm could be produced in many instances by stimulation of the left sympathetic nerve, and that this was

specially likely to take place if the right vagus was stimulated at the same time. Wilson[14] was able to cause A-V rhythm in man by deep breathing or ocular compression at a certain critical period after the administration of atropine. In two cases of heart disease this effect took place without the use of the drug. He suggested that atropine paralyzed the vagal fibers to the A-V node before those to the sinus, so that inhibitory control was removed from it while a considerable amount was still present in the sinus. In the two cases where no drug was used, he attributed the effect to depression of the sinus rhythm. In the present case the rhythmicity of the A-V node was undoubtedly increased as the rate of the heart was slightly greater than normal. It is impossible to state whether there was also a depression of the rhythmicity of the S-A node, although this seems likely as the heart rate did not attain the height which is usually associated with the dose of adrenalin which was given. It seems possible that in this case sympathetic stimulation increased the rate of impulse formation in the A-V node, but at the same time the accelerator effect of sympathetic stimulation on the sinus was neutralized by increased inhibition. The possibility of the latter is suggested by Kahn's observation that the complete heart-block which he obtained in animals, after adrenalin, was abolished by section of the vagi, although in these experiments of course the main inhibitory action was produced in the A-V node.

The fact that the heart was controlled by the A-V node during the greater part of the paroxysms of tachycardia from which this patient suffered, and that A-V rhythm was produced by adrenalin tends to suggest that in him the A-V node was more sensitive than in the majority of individuals.

SUMMARY

Attacks of paroxysmal tachycardia of supraventricular origin are described in an otherwise normal individual. During these attacks the site of impulse formation appeared to change. It is suggested that the heart was under the control of the sino-auricular node for a short period at the beginning of the attack, but that later it responded to the lower part of the auriculoventricular node. The site of stimulus production gradually moved to a higher level in this node as the paroxysm continued. The attacks could be terminated by a deep breath. This patient showed a similar response to atropine as normal subjects, while adrenalin produced nodal rhythm.

I wish to thank Mr. Fraser, the patient, for his cooperation in the investigation, and the Trustees of the Clinical Medicine Research Laboratory for permission to work there. The expenses of the work were defrayed by a grant from the Earl of Moray Research Fund, University of Edinburgh.

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THE TREATMENT OF TRANSIENT VENTRICULAR STANDSTILL WITH BARIUM CHLORIDE*

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BARIUM chloride has recently come into use for the treatment of syncopal attacks in complete heart-block. Its use in cases of transient ventricular standstill has been extremely rare. In fact cases of transient intermittent heart-block are not of frequent occurrence.

Cohn and Lewis[1] in 1911 reported a case of transient heart-block with fits in a woman eighty years of age. There was an interval of eight months' freedom from attacks, with return of attacks followed by complete block eleven months after onset. The patient died seventeen months after onset. Post-mortem studies showed a fibrosis of the auricle and an increase of the connective tissue about the nerves and ganglia of the interauricular septum.

Starling[2] reported a case of transient heart-block due to vagal stimulation in a man fifty-one years of age. The attacks were provoked by swallowing and were abolished by atropine. These attacks continued until complete heart-block ensued.

Wilson and Herrmann[3] reported a case in a coal miner, aged thirty-nine years. Adrenalin was tried in this case with questionable effect. *Barium chloride* was also tried for the first time recorded, but no effect was obtained. The patient died six months later. The authors comment as follows: "The mild nature of the attack at this time was due, first to irregular formation of impulses by one of the lower ventricular centers, and later to the development of auriculoventricular rhythm. A center situated above the bifurcation of the His bundle acting as pacemaker. . . . The irregular formation of impulses by some of the lower ventricular centers appears to be a common phenomenon when periods of ventricular standstill occur in rapid succession. It is possible that mechanical factors associated with the overdistension of the ventricle or with the auricular contraction may play a part in their initiation."

Wilson and Robinson[4] reported a case of transient complete heart-block, with numerous attacks of Stokes-Adams' syndrome. The onset was with pain in the epigastrium. The electrocardiogram showed the usual type in cases of this sort and was similar to the tracing in the case of Wilson and Herrmann.

Lewis,[5] in his *Lectures on the Heart,* reports two cases of standstill

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of the ventricle. The first case, a man of forty-eight, who had an aortic stenosis, had for some years experienced attacks of giddiness followed by a temporary lapse of consciousness. The cardiac mechanism before and after the attack was natural. During the attack the pulse failed for from three to eight seconds. A lapse of five seconds sufficed to abolish consciousness. During the period of standstill, the heart sounds were not heard. The auricles continued to beat at their former rate. Lewis thinks that there was a lesion of the auriculoventricular bundle in this case. In the second case the fits came in groups between intervals of freedom which lasted several months, and on the days on which fits were present there was a partial heart-block of low grade. At post-mortem examination large blood sinuses were found in the auriculoventricular bundle, the tissue having the appearance of a nevus. "It is easy to comprehend how in this instance an added strain, with engorgement of these vessels would bring about cessation of the ventricular beat."

Gager and Pardee[6] report a case in a man of fifty-nine, who at times had complete heart-block with Stokes-Adams' attack due to ventricular stoppage; at other times normal heartbeat with normal conduction time. Digitalis precipitated syncopal attacks in the presence of a complete block. In this case "the evidence suggests that ventricular stoppage was due to variations in ventricular rhythmicity. The changes in form and the duration of the ventricular complex of the electrocardiogram immediately preceding the periods of ventricular standstill are proof of change in the pathways of intraventricular conduction and by inference in the site of the impulse. It is reasonable to suppose that the ventricular tachycardia depresses the irritability of the ventricle and brings about inactivity of one pacemaking center after another until stoppage occurs. Changes in nodal and ventricular rhythmicity appear to be the governing factor in producing standstill while definite vagus and auricular influences are absent in this case."

Dana and Dameshek[7] report a case of intermittent auriculoventricular block, apparently due to a right bundle-branch defect, in a man seventy-six years of age.

McIntosh and Falconer[8] report two cases of Stokes-Adams' disease, transient in character with normal conduction time.

Gosage[9] reports a case of Stokes-Adams' syndrome due to ventricular standstill, and ascribes it to depression of excitability of the ventricle.

Wiltshire[10] reports a case of heart-block, illustrating the behavior of the auricle during prolonged ventricular standstill.

Carter and Dieuaide[11] report a case of a man, seventy years old, who had attacks of syncope brought on by straining at stool, by lying on his left side, and by abdominal distension. Attacks lasted

from one to two minutes with a regular pulse of from sixteen to twenty per minute. Though pressure on the vagi had little effect, 2 mg. of atropine administered during an attack of complete heart-block caused a reversion to normal mechanism.

These authors have reviewed the literature of transient heart-block very completely and have summarized the cases reported up to that time. It is worthy of note that most instances previously reported have occurred in elderly individuals, whose previous history throws no light on the course of the condition. In one case (Lewis[5]) rheumatic infection could have been the etiological basis. In another case (Wilson and Robinson[4]) acute infection of unknown nature may have been the cause. The most significant finding seems to be a general vascular sclerosis.

"The reported cases[11] of abrupt transient auriculoventricular dissociation are rather few in number, but the data available suggest that they are usually not fundamentally different from the other causes of heart-block. Of eleven patients, nine showed transient auriculoventricular dissociation, seven are known to have developed complete block, and nine died in a short time. In the only instances in which the heart was examined, definite lesions of the auriculoventricular bundle were found. As a rule atropine was ineffective in relieving the block during dissociation.

"In the light of modern physiochemical hypothesis of the nature of the conduction process, it may be inferred that there exists a large reserve in the conducting capacity of the auriculoventricular bundle which may be seriously encroached upon before conduction is measurably impaired. A few intact fibers may under favorable circumstances serve to carry on conduction processes normally, while some subtle local circulatory deficiency or temporary increase in vagal action may result in failure of the few remaining fibers to function. This point of view is also helpful in understanding the reported instances of lesions affecting the auriculoventricular bundle in which no heart-block has been recorded. The warning of Lewis may therefore be repeated, that the atropine test is not necessarily decisive as to the pathogenesis of heart-block and that when such an apparently marked vagal action as release from complete heart-block is encountered this response may take place because of the existence of an anatomical lesion which is already dangerously near causing permanent dissociation."

The nature or physiology of ventricular stoppage as appears from analysis of the above cases in the literature is seen to be a varied one. Experimental work on animals (dogs) by Erlanger and Hirschfelder[12] directs attention to four ways in which ventricular stoppage may be produced:

1. By inhibition of the auricles through vagus stimulation, while the conduction system is intact.

2. In the presence of partial heart-block, by an increase in auricular rate, which produces fatigue of the auriculoventricular bundle.

3. By compression of the bundle sufficiently to produce complete block.

4. In the presence of total heart-block by the sudden cessation of fairly rhythmic ventricular stimulation.

It is evident that any of the several mechanisms discussed by Erlanger and Hirschfelder may be operative in different patients.

The literature reviewed above shows that barium chloride has not been used in transient ventricular standstill, except in the case of Wilson and Herrmann. We now give the details of our observations.

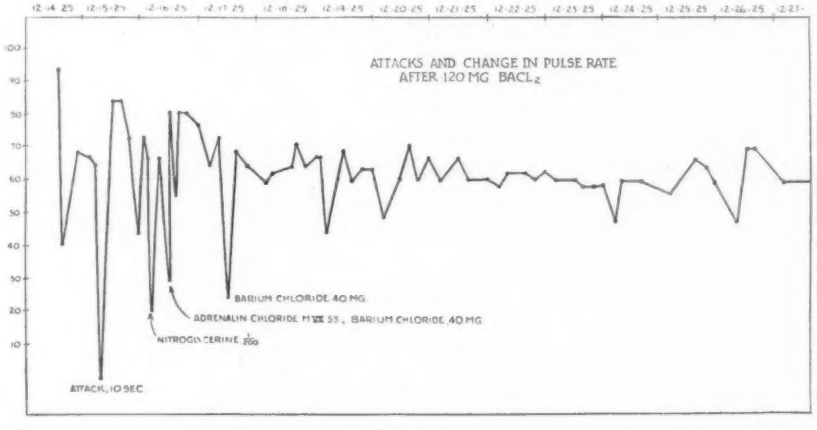

Fig. 1.

REPORT OF CASE

Our patient, Mrs. Y., aged fifty-five years, was admitted to the gynecological service on June 30, 1925, complaining of fainting spells. She was perfectly well until three months before admission (April, 1925), when suddenly while sitting in a chair, she felt a pricking sensation in the feet and then fainted. She regained consciousness and felt perfectly well. From this time the patient began to have spells of fainting daily and at times several times a day. They occurred during rest, while standing, or when walking. The fainting spells lasted only a short time, the patient being entirely unaware of the onset and unconscious during the attacks. There were no other complaints except constipation. Past history was negative except for measles in childhood.

Physical examination was negative except for a fibroid of the uterus. Pulse on admission was eighty-four and was never below seventy-two during this stay in the hospital.

She was operated upon on July 3, 1925, and a large calcified fibroid of the uterus was removed. She was discharged from the hospital July 15, 1925, well.

On December 14, 1925, patient reentered the hospital on the medical service of Dr. Solomon Strouse complaining of dizziness, ringing in the ears, blurring of vision, swelling of extremities, and stomach trouble.

Since the date of her dismissal from the hospital, July 15, to the date of readmission, December 14, she had been well, complaining only of occasional dyspnea on exertion. She had no attacks of dizziness, or fainting spells, up to the day previous and the day of admission, at which time she had many repeated attacks. The attacks are described as dizziness followed by unconsciousness, after which she suddenly recovered and felt well. These spells lasted for a "minute," but on the day of admission she had them continuously. The attacks cannot be definitely related to any gastrointestinal disturbance, but the patient said she felt worse when constipated. Patient stated that early in the beginning of her attacks she was aware of the onset by "dizziness" and would cry out. At the time of admission the "crying out" did not occur. She attributed her entire illness to an attack of influenza two years previously.

On admission, December 14, 1925, pulse was 88, and five hours later the nurses' records showed a pulse rate of 40 (Fig. 1), with the notation that patient's pulse was intermittent and that she looked faint and was dozing. On the following morning one of us examined the patient and made the following notation: "Examination reveals a very pale woman, who gets 'dizzy' spells, and who becomes unconscious during examination. The pulse rate when first examined was sixty-four. Almost immediately, there was a cessation of the pulse at the wrist and also at the apex. This lasted for about ten seconds." A diagnosis of Stokes-Adams' syndrome was made. Recovery from attack occurred, and patient was comfortable through the day. Small doses, 1/200, of nitroglycerin were given. The heart borders measured 3.5 cm. to right of midsternal line and 12 cm. to the left of midsternal line. A faint systolic murmur was present at the apex. The blood pressure was 178/80. Urine was negative on repeated examinations. The Wassermann test was negative. The x-ray film showed a bulging and tortuosity of the aorta.

The next morning (December 16) the pulse was much slower; nine beats were counted, then there was an interval of twenty seconds during which the patient became very pale and unconscious, and her left arm fell to one side. The pulse returned suddenly with a severe vomiting spell. There were several spells of vomiting during the day. The patient was semicomatose during the greater part of the day, moaning constantly. The pulse chart for December 16 is as follows:

8:00 A.M.	pulse at wrist 20.	Nitroglycerin 1/200 at 8:30, 845, 9:00. Morphine sulphate, gr. 1/6, at 9:30 A.M.
2:00 P.M.	" " " 35.	Tincture of digitalis, m. xv.
3:00 P.M.	" " " 29.	Adrenalin chloride, m. vii.
5:00 P.M.	" " " 56.	Tincture digitalis, m. xv, one dose.
6:00 P.M.	" " " 80.	Barium chloride, 40 mg.
7:30 P.M.	" " " 80.	

The pulse remained normal through the night. Two doses of barium chloride were given on the following day. All medications were discontinued for ten days, when ammonium bromide was started. The patient was much improved. The pulse was regular. An electrocardiogram on December 17 showed normal sinus rhythm. The patient continued well, without any attack until January 10, 1926. The immediate result following the administration of but 120 mg. of barium chloride was most striking. No attacks were noted for a period of twenty-five days. The pulse was as low as forty-eight and as high as eighty, but the general average was about sixty-two.

On January 10, 1926 at 10:55 A.M., the patient stated that she was not feeling well (Fig. 2). The pulse was of normal rate and rhythm. The tones were clear, except for a systolic murmur at the apex. One of us, who had made these observations, then started to examine the next patient, and in so doing noticed that

Mrs. Y. was paling and flushing. Examination now showed that the pulse had suddenly dropped to forty. The heart stopped beating for ten seconds. Then the heart beat twelve beats, a pause of eight seconds, then returned to a rate of thirty-six, then a pause again, and a recurrence of the heart tones with a rate of forty. During attack the patient's face was of a deathly pallor, and when pulse returned, the face would become flushed. Associated with the pallor, there was a marked tremor of the left forearm.

11:00 A.M.	pulse at wrist 35.	Adrenalin chloride, m. vii, given at 11:15.
11:15 "	" " " 34.	
11:20 "	" " " 50.	
11:45 "	" " " 43.	
12:00 M.	" " " 50.	Barium chloride, mg. 40.
1:00 P.M.	" " " 52.	
4:00 "	" " " 68.	Barium chloride at 4:00 P.M.
8:00 "	" " " 72.	

This record, together with our first experience, directed our attention to the transient effects of adrenalin chloride as contrasted with barium chloride. Though adrenalin chloride increased the pulse rate, clinically the patient was not improved.

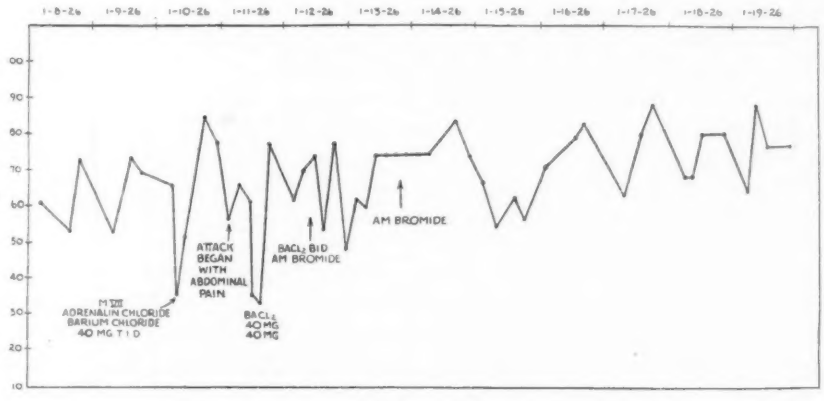

Fig. 2.

The following day, January 11, at 10:30 A.M., the pulse was regular, of good quality, and the tones at the apex were distinct. Patient felt comfortable and wanted to get out of bed. At 12:40 P.M. an attack began similar to that of the previous day. Barium chloride (40 mg.) was given at 1:30 P.M. At the onset of the attack the pulse rate was 60; at 1:30 it was 36; at 2:50 the pulse was still 34. At 4:20 the resident physician was summoned because of another attack. Pulse was 32, extreme pallor, tremor of hands, and at times of the body were present. Barium chloride was given at 4:30, and an electrocardiogram was taken. At 5:20 coincident with the application of heat externally and hot tea by mouth, the pulse returned to 56. Patient seemed better, talked, and felt relieved. During this attack, abdominal pain was complained of. At 7 P.M. the pulse was 72 and at 8 P.M., 76.

Barium chloride was now continued three times a day (40 mg.) for one day and then discontinued. The patient's pulse was regular and ranged from 54 to 80 for eleven days. During this time the patient had received ammonium bromide, grains xv, three times a day, also atropine sulphate, grain 1/100, three times a day for six doses. On January 19 pressure was exerted on the vagi. Pressure on the right vagus had no effect. Pressure on the left vagus had no effect. Pressure on both vagi elicited two attacks. The patient suddenly became pale and un-

conscious, and the pulse became imperceptible. As patient was becoming conscious, both arms were raised convulsively. Electrocardiographic tracings were taken. At the end of the test atropine, grain 1/50, was given. Pulse became faster than it had been at any time since hospitalization.

The twelfth day after last administration of barium chloride (January 23), the patient had a mild attack which was relieved by heat, hot brandy, and an injection of morphine sulphate, grain 1/6, and atropine sulphate, grain 1/100. Atropine, grain 1/100, was again given twice a day for three days, the pulse ranging from 80 to 54. During this period no barium was given. On January 26, at 6:15 A.M., an attack began with irregular and slow pulse, and patient complained of pressure and pain over chest. Atropine, grain 1/100, was given with no effect. Barium chloride (40 mg.) was given at 6:45 A.M., and there was a decided but gradual increase in rate and quality of pulse. At 8 A.M. pulse was 48. At 10 A.M. it was still 48 and patient complained of pain in the abdomen. Barium chloride was again given at 12:50 P.M. At 1:45 P.M. pulse was 38, and hard to get at wrist or apex; marked pallor of face but hands and body were warm. Atropine, grain 1/100, was again given, and in fifteen minutes the pulse was 48. At 6:25 P.M. barium chloride was given; at 6:35 the pulse was 36, irregular, and of poor quality; at 6:55 P.M. the pulse was 44 to 46, and of good quality; the patient was warm and felt better. At 9:45 P.M. pulse was 80, and the next morning pulse ranged from sixty to seventy-two.

Barium chloride was now continued, 40 mg. three times a day, for two days, then reduced to once a day for one day, and then twice a day for two days. During this time the pulse was regular and ranged between 60 and 76, and there were no attacks. On January 31, five days after last attack, on the morning of which the patient received no barium, an attack occurred. At 10:45 A.M. the pulse was 36. Heat and atropine, grain 1/50, gave no relief, but gradually the pulse increased to 42. Barium chloride was given at 1:40 P.M., the pulse was 52, and at 2:30 P.M. the pulse 48, but more regular, and patient stated she was better. At 8:00 P.M. pulse was 50, and patient was comfortable. The following day, February 1, barium chloride and morphine sulphate, grain 1/6, were ordered twice a day and continued for three days. Attacks occurred on February 2 and were severe and numerous, and were not relieved by atropine. The pulse rate remained about 30 until 5:00 P.M., when it was 68. On this day, in addition to attacks, patient complained of pain in the epigastrium. On examination no definite findings except tenderness over the epigastrium were present. Late in the afternoon patient vomited thirty-six ounces of brown fluid and felt better, but no change in pulse rate was noted for two hours. The next morning her pulse was 72, and continued so for the day. Barium was stopped on February 3, and on the following day the patient had another attack. From February 4 to 11, although the pulse was at times as low as 28, the patient on the whole was comfortable and was able to undergo a complete x-ray examination of the gastrointestinal tract. On February 9, patient received potassium iodide, m. v, three times a day, and this was continued for two days.

On February 11 the following notation was made: "Patient has been having rather frequent attacks—several last night and one this morning. At 10:30 A.M. the pulse was regular, about 50, when she suddenly began to groan, and pulse was imperceptible. There was one pause of eleven seconds and one of six seconds. After recovery from the former, the pulse became rapid and gradually slowed down. At 10:33 the patient was given barium chloride, 40 mg. Shortly after this the pulse became very irregular with frequent short pauses. Patient groaned continually and seemed semicomatose. This lasted for fifteen minutes."

Before onset of first attack described, the patient complained of pain in the epigastrium. She also complained of peculiar pain in the left lumbar region

preceding attacks. The offset of attacks was manifested by tremor of lips and hands and increase in groaning. The rate at wrist was 68. This was quickly followed by another attack. An attempt to aspirate the gastric contents caused marked retching but no apparent relief.

From February 12 to 20 (Fig. 3), the pulse as described by the nurses' chart showed daily variation from 26 to 34. Sometimes transient relief was obtained by nitroglycerin, but notations of repeated attacks occurred daily for a period of eight days. Thus, on February 14 the pulse was slow and regular, with a rate of 36. Pulsus bigeminus was noted at the apex by one of us. On February 15 a severe attack was apparently temporarily relieved by nitroglycerin. On February 19 the pulse was full, bounding, slow, and irregular, with few pauses of from two to three seconds. Sounds at heart again indicated a bigeminus at times. February 16 potassium iodide, m. v, three times a day was again started and was discontinued three days later. Ammonium bromide was started on the sixteenth of February, and continued during her stay in the hospital. Barium chloride, mg. 40, four times a day, was started February 20, and continued during her stay in the hospital.

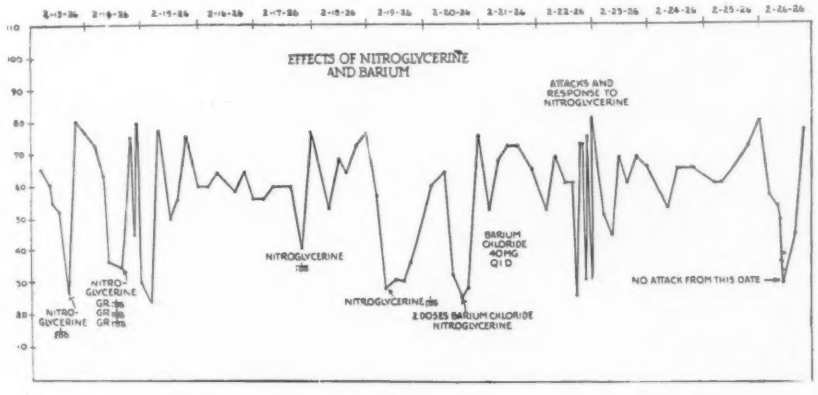

Fig. 3.

Patient had severe attacks two days later, although she had been receiving barium chloride. Nitroglycerin gave temporary relief. Another attack occurred four days later, a few hours after the patient had been up and about. This attack also seemed to be relieved by nitroglycerin. There were apparently daily attacks up to the fifth of March. During these days the pulse varied from 54 to 72, during the attacks going as low as 28. From March 5 to the date of discharge (March 22), there were no attacks. Her condition on discharge was good. Patient flushed easily. The left border of the heart was 11.5 cm. and the right border, 2.5 cm. from midsternal line. Tones were clear, regular, and distant. Pulse rate was 74. Blood pressure, 135/85. Lungs were negative.

Patient was seen at frequent intervals at the dispensary, and at home by one of us from the date of discharge up to her final entrance to the hospital. Barium chloride, 40 mg. three times a day, was taken continuously during this whole period. Though there were various minor complaints, such as dizziness, constipation, and pain in the epigastrium, no attacks were noted. On December 5 pressure was made on the vagi while the patient was being observed with the electrocardiograph, with no apparent change in pulse.

On December 28, patient developed another attack. This was characterized by slowness of pulse, feeling of discomfort, and pallor. Patient went to the hospital three days later. During this time she had attacks with slowness of pulse, and

absence of beats for from three to five seconds. There were no attacks of unconsciousness or convulsive seizures. Tincture of digitalis was given in addition to barium chloride, and this was followed by atropine. During the first few days at the hospital the attacks were relatively few. An electrocardiogram taken January 4, 1927, showed a sinus rhythm, and a ventricular complex similar to those in previous electrocardiograms. On January 6 the patient had an attack, the pulse dropped to 32, but there were no subjective symptoms. Following atropine the pulse rate was 92, and remained regular for approximately four hours. The pulse then dropped to 20, again returned to 88 following atropine. In the evening the pulse dropped to 34 and remained slow up to the time of her death, January 8. Adrenalin chloride and atropine administered January 7 and at the time of death had no influence. Post-mortem examination was not permitted.

ELECTROCARDIOGRAMS

The electrocardiogram taken on the day after entrance (December 15, 1926) (Fig. 4) shows repeated short attacks of ventricular standstill interrupted by left ventricular extrasystoles. At times during

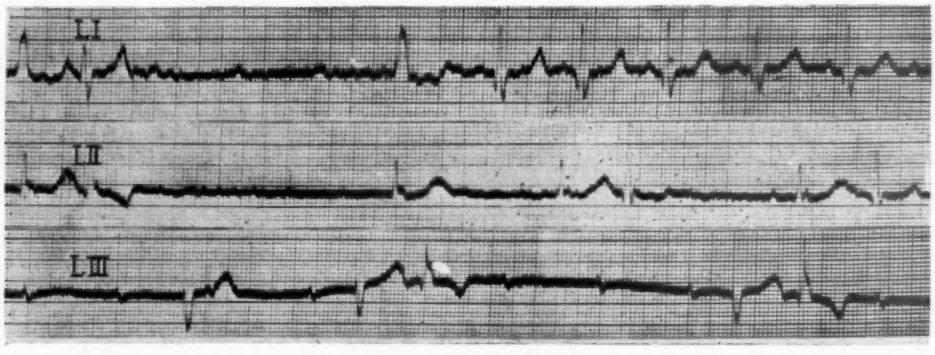

Fig. 4.—December 15, 1925.

fairly long periods the only ventricular beats are extrasystoles; at other times extrasystoles are succeeded by supraventricular complexes. In Lead I there is a stretch of at least six beats of regular sinus rhythm. In Lead III the supraventricular complexes following the extrasystoles are wide and notched.

Electrocardiogram taken December 16, 1925 (Fig. 5), the day when patient was having continual attacks which were finally relieved by barium chloride, shows the longest periods of standstill. Early in Lead I there is a standstill of 16⅗ seconds interrupted by a left ventricular extrasystole with a high distant T-wave, followed by six beats of supraventricular origin with high distant T-waves. The rate of each of these beats is different, varying from 40 to 100 per minute. At times the R-wave is inscribed upon the preceding T-wave. These beats are followed by the second standstill, 17⅕ seconds in length, interrupted by an extrasystole identical with that of the first standstill, which in turn is followed by five supraventricular beats of the

same character and with the same irregularity of rate as above. These are followed by the third standstill, the offset of which is not recorded.

In the second lead, three more standstills are recorded, the beginning of the first and the offset of the third are missing. The second is 16⅗ seconds in duration. Here again each offset is due to an extrasystole, followed respectively by eight and six supraventricular beats with high T-waves and of an irregular rate varying as in Lead I, from

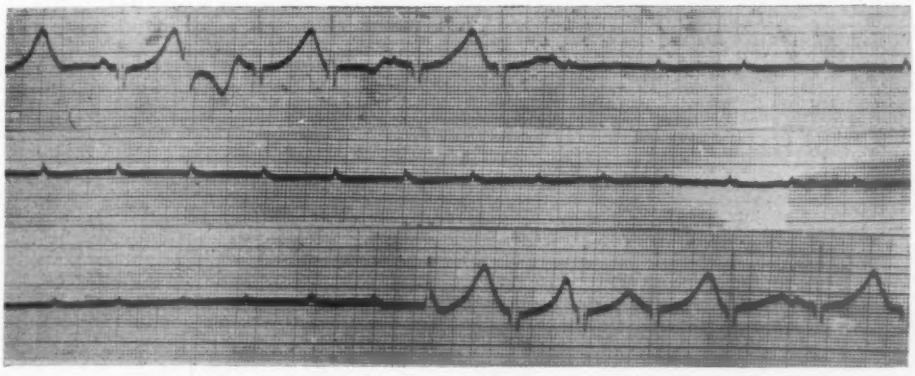

Fig. 5.—December 16, 1925. Lead II, strips 1, 2, and 3 continuous.

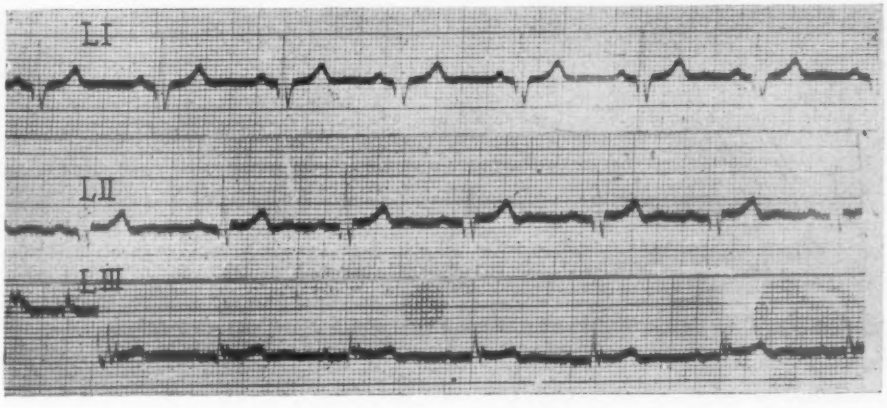

Fig. 6.—December 19, 1925.

40 to 100. The beginning of Lead III differs somewhat from the others; at first three broad, notched, supraventricular beats are recorded, followed by one large ventricular or nodal extrasystole which is followed by a standstill of 4⅗ seconds, interrupted by one large extrasystole; then a standstill of 16⅘ seconds. This is ended by a complex partaking somewhat of the form of the supraventricular complexes, followed by a large extrasystole of the same nature as the two above, followed in turn by four broad, notched supraventricular beats. The film ends in a standstill of 13⅕ seconds and several ventricular beats.

It is noticeable that at the onset of the standstill the auricular rate is slow, about 68 per minute, gradually increasing in the longer pauses to a rate of 100. According to Lewis[13] during periods of ventricular silence, it is customary for the auricular rate to accelerate.

On December 19, 1925 (Fig. 6), the records show a normal sinus rhythm, following the administration of barium chloride. The tracing is normal except for broad notched QRS complexes in Lead III.

On January 11, 1926, the second series of attacks is recorded. In Lead I there are numerous short attacks of standstill interrupted at times by single normal beats or by single nodal extrasystoles. The longest period of standstill is 5⅕ seconds. The auricular rate is 100 per minute. The T-waves are not abnormally high.

Leads II and III are much the same as Lead I. The T-waves are higher than in Lead I but not as high as those recorded on December

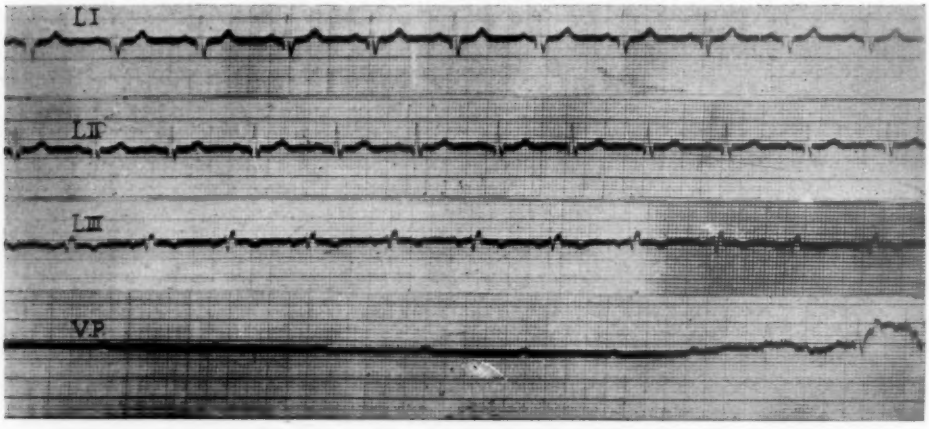

Fig. 7.—January 19, 1926. Strips 1, 2, and 3, three leads before pressure; strip 4, standstill due to pressure on both vagi.

16, 1925. There are some periods of standstill interrupted only by extrasystoles (ventricular escape?). The attacks finally end with the resumption of the normal sinus rhythm toward the end of the day after the administration of barium chloride and the application of heat. The rate is 70 per minute, the P-R interval 0.18 second, the same as the P-R interval during the time of the attacks. The auricular rate at the time of standstill is approximately 100 per minute. The QRS complex in Lead III is broad, low, and splintered, with a deep broad S-wave. Complexes in all leads resemble normal beats during standstill, except that the T-waves are of normal height.

On January 19, 1926 (Fig. 7), pressure was exerted on the vagus. Preceding pressure all leads show normal sinus rhythm, with a rate of 70 and a P-R interval of 0.18 second. The QRS in Lead III is broad, splintered and low, and T is inverted. Pressure on the right and left vagi separately had no effect. Pressure on both vagi caused a stand-

still of 8⅗ seconds, ending in a convulsive tremor of the hands which obscures the tracing. Release shows a return to the normal sinus rhythm at a rate of 107. This quickly slowed down to 70 when pressure again brought on an attack similar to the above. At another time distinct sinus arrhythmia was brought on by pressure, the rate varying from 50 to 75.

The electrocardiogram taken on July 28, 1926, after patient had been on barium chloride continually for five months, shows sinus rhythm—a rate of 62 per minute, P-R interval of 0.18 second, a notching of QRS complex, and occasional auricular extrasystoles.

On December 8, 1926, after eleven months of barium chloride, pressure was again exerted on the vagi without effect. The tracings taken

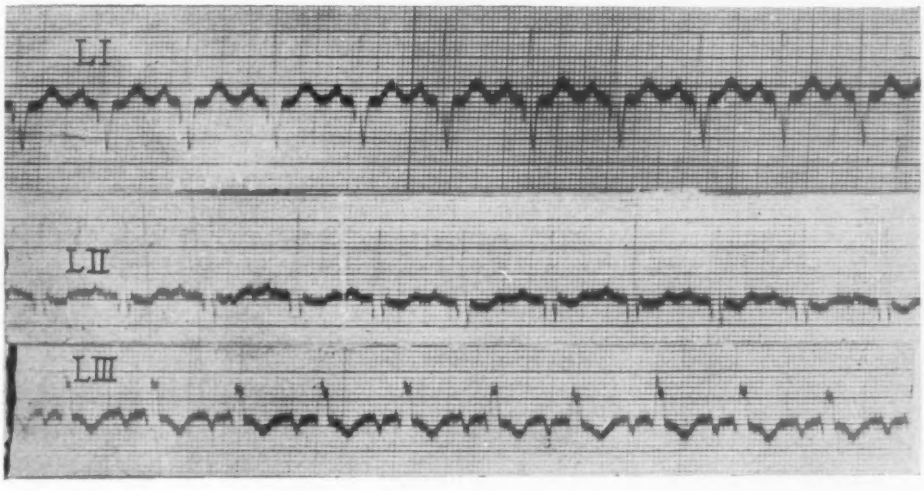

Fig. 8.—January 4, 1927.

on this date show normal sinus rhythm of the usual character for this patient. P_3 is inverted, T_3 is iso-electric, and QRS_3 is broad and notched.

About three weeks after this, the attacks recurred, and the patient reentered the hospital. On January 4, 1927 (Fig. 8), four days before death, the tracings still show a sinus rhythm, rate 88, P-R interval 0.2 second, inverted P_3, and T_3, and a broad notched QRS_3.

The last electrocardiograms were taken on January 7 (Fig. 9), one day before death, when the patient was having continual attacks and in much the same condition as on her previous entrance in December, 1925. The tracings show a complete absence of sinus rhythm. The auricles are beating regularly at a fairly slow rate. Ventricular beats appear at frequent intervals singly, in pairs, or in series of three or four beats. The ventricular complexes appear to arise in five or six different foci, mostly ventricular but a few nodal.

The electrocardiograms show a transient stoppage of the ventricles, in all of which except those taken one day before death, beats arising

in the sinus appear singly or in series. These beats are of the same character as those appearing when there is no irregularity, and the P-R interval is at no time prolonged, being 0.18 of a second. Very often the standstill is interrupted only by single extrasystoles, an apparent escape of the ventricle. In the final electrocardiogram there is a marked variation of ventricular complexes, due, we think, to the attempt of the ventricle to take up its work again under great difficulties, appearing at times like a delirium cordis, bordering closely on ventricular fibrillation.

At all times the QRS in Lead III is broad and notched, probably indicating an arborization block and involvement of the fibers of Purkinje.

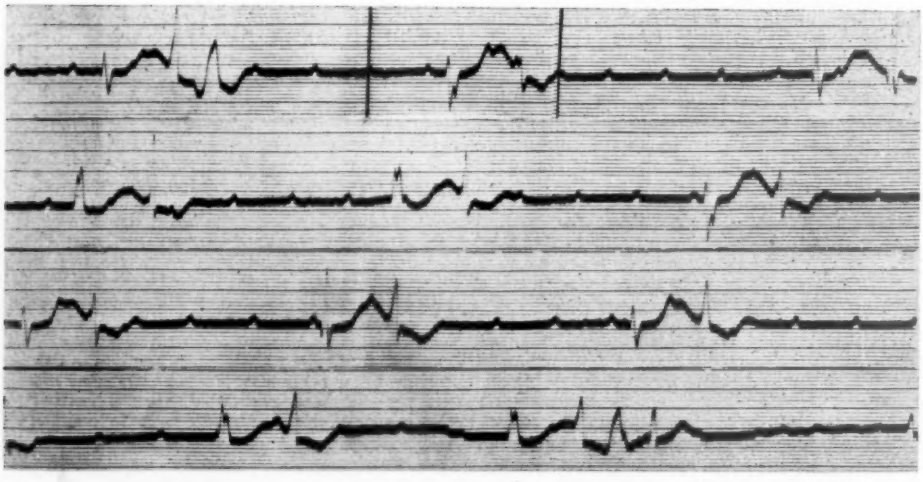

Fig. 9.—January 7, 1927. Strips 1, 2, 3, and 4 continuous.

SUMMARY OF TREATMENTS

In the first attack, adrenalin subcutaneously apparently gave no immediate relief of symptoms, although the pulse rate was temporarily increased. One hundred and twenty milligrams of barium chloride apparently gave relief and changed the pulse rate, so that no attack occurred for a period of twenty-five days. Attacks recurred after moderate exertion.

In the second attack, adrenalin, m. vii, apparently gave the same result as in the first attack. Barium chloride, 80 mg., was sufficient only to relieve the patient for the night; the next morning, another, the third attack occurred.

In this attack two hundred milligrams of barium chloride during and following the attack resulted in freedom from attacks for eleven days. Atropine sulphate, gr. $\frac{1}{100}$ three times a day, and ammonium bromide, gr. xv three times a day, were given, then a mild attack

came on. Atropine sulphate, gr. $\frac{1}{100}$ twice daily, was given for three days with no effect. A severe attack then followed. Atropine apparently gave no relief, but after barium was administered the pulse increased to within normal range.

Two hundred eighty milligrams of barium prevented attacks; atropine, $\frac{1}{50}$ grain, failed to improve an attack.

Vomiting seemed to relieve the patient during an attack; 120 mg. of barium in a period of three days failed to prevent an attack from occurring the day barium was stopped, but during the day that it was readministered, no attacks occurred.

For a period of seven days, during which the patient received no medication except potassium iodide for three days, no attacks occurred.

Nitroglycerin, gr. $\frac{1}{100}$, gave transient relief from unconsciousness at times, but there was no change in pulse rate for a period of eight days.

Following the daily administration of barium chloride, except for those transient attacks during the first days of administration, no attacks or changes in pulse rate from normal were noted for a period of one month to date of discharge. Previous to the continuous administration of barium, potassium iodide was given for a period of three days.

A total of 6,320 mg. of barium chloride was administered to the patient while in the hospital.

At times some of the attacks seemed intimately associated with gastrointestinal discomfort, and in one instance in particular, vomiting relieved the attack. A complete study was made of the gastrointestinal tract, and no evidence of organic trouble could be found.

The clinical and roentgenological findings supported the clinical diagnosis of a senile aortitis.

From an analysis of our case, we undoubtedly were dealing with a transient heart-block in which there was diminished irritability of the ventricle. It is very likely also that the vagus played a rôle, for we were able to obtain a definite attack by pressure on both vagi. The onset of attacks were also often attended with gastrointestinal disturbance, and it is conceivable that reflex vagus inhibition may have originated from this source.

DISCUSSION OF TREATMENT

Hirschfelder[14] divides the treatment of Stokes-Adams' disease into three phases as follows:

1. To bring about retrogression of the lesion in the bundle.

2. To remove as many factors as possible which tend to increase the block.

3. To increase the irritability and rhythmicity of the ventricular muscle, so as to shorten the periods of stoppage and to increase the rate of the ventricles.

The first phase of this treatment is directed, of course, to luetic lesions of the bundle. In many instances this results favorably. Herrmann and Ashman[15] direct attention to severe symptoms which seem to have been brought on by the "mixed antiluetic treatment," and attribute these disturbances to the depressing effect of potassium salts, producing or increasing an already present conduction disturbance. In our case potassium iodide apparently produced a similar effect.

Atropine has been known to be an effective therapeutic agent in the relief of heart-block, which is vagal in character. However, Hirschfelder states that in the cases in which the lesion itself cannot be made to subside, especially in cases in which the block is a partial one, it may be possible to bring temporary improvement by removing the tonic action of the vagus through atropine. In our case atropine had no apparent effect, except during the attack just before death. It is significant, however, that pressure on both vagi was effective in producing attacks of heart-block. This supports the observation that vagal influences may enhance the organic lesion, although the vagus itself may not be the exciting cause of heart-block.

Adrenalin.—The use of adrenalin in the treatment of syncopal attacks that occur in Stokes-Adams' syndrome was suggested by Phear and Parkinson.[16] The action of adrenalin is twofold: it accelerates the heart by direct stimulation, and inhibits it through the vagus center. The net result of this balanced mechanism, according to Meek and Eyster,[17] is a decrease in pulse rate. According to Phear and Parkinson, experimental work on animals indicates that adrenalin accelerates the ventricle as well as the auricle, even when complete block is present. They have reviewed the clinical and experimental literature and conclude as follows:

"The clinical evidence shows that it is possible for partial block to be reduced and for even complete block to be abolished by the subcutaneous injection of adrenalin, though often it fails to modify conduction as one might expect from the nature of the pathological lesion usually present. An increase in ventricular rate is usually obtainable despite the block. This alone is sufficient ground for an extended trial of adrenalin in Stokes-Adams' attack, when the immediate cause of loss of consciousness is usually extreme ventricular slowing and standstill."

Herrmann and Ashman,[15] likewise Levine and Matton,[18] draw attention to the fact, that when adrenalin is given subcutaneously, it can hardly act until several minutes have elapsed and therefore can have no immediate effect on an attack if the patient is in syncope at

the time. Furthermore, at the moment of complete standstill of the heart there is no appreciable blood flow, and although slight local diffusion may occur when adrenalin is injected subcutaneously, none of it could reach the heart if injected intravenously. It is necessary to inject the heart directly in such cases, according to these authors, and Herrmann speaks of it as the drug of choice.

Nitroglycerin.—A study of the pulse charts shows that nitroglycerin was efficacious in producing an acceleration of pulse rate in the short period of approximately one-half to three hours. However, it had no effect on preventing the recurrence of attacks. It is suggested that nitroglycerin may produce a vascular dilatation, and this in turn may stimulate an automatic increase in pulse rate to maintain the circulation. In our case nitroglycerin seemed to act better when the patient was under the influence of barium chloride.

Barium Chloride.—Rothberger and Winterberg[19] showed that barium and also calcium tend to increase the irritability of the ventricle, as shown by the occurrence of ventricular extrasystoles at first singly and then in shorter and longer runs of tachycardia.

Von Egmond[20] demonstrated that barium chloride exerts the same influence after complete traumatic idioventricular block.

Cohn and Levine[21] were the first to use barium chloride for the prevention of the recurrence of syncopal attacks that accompany ventricular standstill. In a series of three cases apparently good results were seen while the drug was regularly administered. Two of their patients died ten and seven months respectively, following discharge from the hospital. The third patient was at work one year after the report. The first patient who died received 240 mg. of barium chloride in six doses within forty-eight hours and was given no more. After discharge from the hospital the attacks began to recur, and calcium lactate in 1 gm. doses was given three times daily, but sudden death occurred ten months after the course of barium chloride. The other patient received 810 mg. while in the hospital, and after discharge was given barium chloride, 30 mg. three times a day, for ten days. It is of interest to note that in these cases complete heart-block was present. Our case was one of definite transient heart-block, and we feel that probably the indication for barium is greater in such cases, especially if we consider that such attacks are due to a lowered excitability of the ventricle.

Heard, Marshall, and Adams[22] reported a case of partial heart-block that was treated with barium chloride. This patient received a total of 5 grams over a period of three months, during which time they observed neither favorable nor unfavorable effects. Ectopic ventricular impulses did not appear. The attacks continued with increasing frequency and in one of these the patient died.

Herrmann and Ashman[15] reported favorable results with barium chloride in a case of transient complete heart-block and in another case of complete heart-block with syncopal attacks. They believe that the intermittent use of barium chloride is likely to prove dangerous, principally because the patient is likely to become careless and neglect his treatment, while if he is impressed with the fact that his life depends on the presence of a constant minimum concentration of the drug in his heart muscle to keep his idioventricular pacemaker irritable, he will cooperate more conscientiously. They have used doses as high as 50 mg. four times daily over a long period without detrimental effect.

Our experiences with barium would tend to corroborate the opinion and experience of Herrmann and Ashman. Our patient on discharge from the hospital had received approximately seven grams of barium chloride without any harmful effects. She continued to take 40 mg. three times a day from March 22, 1926, to a few days before death, January 7, 1927. It may, however, with some degree of justice, be assumed that transient heart-block may disappear per se independent of medication, as is attested by the story of our own case, in which the attack occurred early and the patient was free of symptoms for a period of six months. There have likewise been reports of patients who have lived for a number of years without treatment. However, attention is directed to our inability to produce heart-block by pressure on the vagi after the continuous administration of barium chloride, whereas we were able to produce heart-block before its administration. Apparently the barium concentration in cardiac muscle was sufficient to prevent the production of cardiac standstill by pressure on the vagi. Attention is directed to the summary of the cases of transient heart-block reported by Carter and Dieuaide,[11] who find that the majority of these develop complete heart-block before death. In our case no complete dissociation developed. The establishment of a normal sinus rhythm, and in particular the cessation of all attacks would justify the continuous administration of barium chloride.

SUMMARY

1. Barium chloride administered early in the course of the disease, even in as small daily amounts as 120 mg. was sufficient to prevent an attack for a period of twenty-five days.

2. The continued administration of barium chloride resulted in complete restoration of normal sinus rhythm for ten months.

3. The continued administration of barium chloride, 40 mg. three times a day, was not associated with any deleterious effects.

4. Our experience confirms the idea of Herrmann and Ashman that barium chloride should be given continuously.

5. Adrenalin injected subcutaneously gave no permanent relief.

6. Atropine did not relieve or check the syncopal attacks, except in two instances just before death.

7. The administration of potassium iodide in small doses seemed to aggravate the frequency of attacks.

8. The studies pursued would indicate that the etiological factor in this case was associated with a depression of irritability of the ventricle, and that the vagus apparently through stimulation via the gastrointestinal tract also played a rôle.

9. The patient died without developing complete heart-block. We believe that the lesion was in the ventricle below the bundle of His, possibly at the junction of the Purkinje fibers and the ventricular muscle.

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ANEURYSM OF THE LEFT VENTRICLE*

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THE designation of any cardiac enlargement as aneurysm by the early writers on cardiac pathology has led to much confusion in the historical research of this subject. The master clinician, Corvisart, further propagated this erroneous conception by differentiating active aneurysm of the heart (hypertrophy) from passive aneurysm (dilatation). To Matthew Baillie is attributed the restriction of the term to a partial dilatation of the heart wall. His classical description[1] of a specimen from John Hunter's museum follows:

"It sometimes happens, although I believe very rarely, that the heart becomes aneurysmal. This disease consists in a part of it being dilated into a pouch, which is commonly more or less filled with coagulated blood. Of this disease I have seen only one instance. The apex of the left ventricle was dilated into a pouch large enough to contain a small orange, was much thinner than in the healthy structure, and was lined with a thick white opaque membrane. There was hardly contained in it, any coagulated blood; but the quantity of the coagulated blood depends commonly on the size of the bag.

"This disease most commonly arose from the muscular structure at the apex of the ventricle becoming weaker than in any other part, so that when the ventricle contracted upon the blood it was pushed against the weakened part, which was not fully able to resist its impetus, and therefore was gradually dilated. Had the strength of the apex of the left ventricle been in due proportion to that of the other parts, it is impossible that the aneurysmal swelling should ever have taken place."

A considerable literature on cardiac aneurysm has accumulated since that time, but the most notable contributions to our understanding of the subject have dealt with the fundamental studies of the coronary circulation and the experimental results of its occlusion. Interference with the nutrition of the myocardium has long been recognized as the background of aneurysmal dilatation. Coronary sclerosis, with or without thrombosis, supplies the pathological equivalent of the experimental ligation studies of Smith[2] and others. Particular importance is attached to these observations because of the close parallelism of their results to the clinical course of coronary occlusion. The time factor in this occlusion has been proved to bear an important relation to survival from the accident. The slower the occlusion, the greater the opportunity for collateral circulation and the maintenance of an adequate nutrition.[3] The smaller the vessel obstructed, the less the danger from such an occlusion. Admitting an ultimately complete interference with the circulation of a given segment of the

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myocardium, the slower the rate of obstruction, the greater will be the opportunity for resorption of the necrotic muscle and for its replacement by fibrous connective tissue. The descendens branch of the left coronary artery is the vessel most commonly occluded. Hence, its supplied myocardium, the anterior aspect of the left ventricle, including the cardiac apex, a small portion of the adjacent right ventricle and of the interventricular septum, would undergo infarction upon interference with nutrition.

As was first pointed out by Hirsch and Spalteholz,[4] a rather smaller area of infarction than is anticipated occurs by reason of the collateral circulation. As a rule, the cardiac apex or the anterior surface of the left ventricle, at least, becomes involved in a myomalacia or

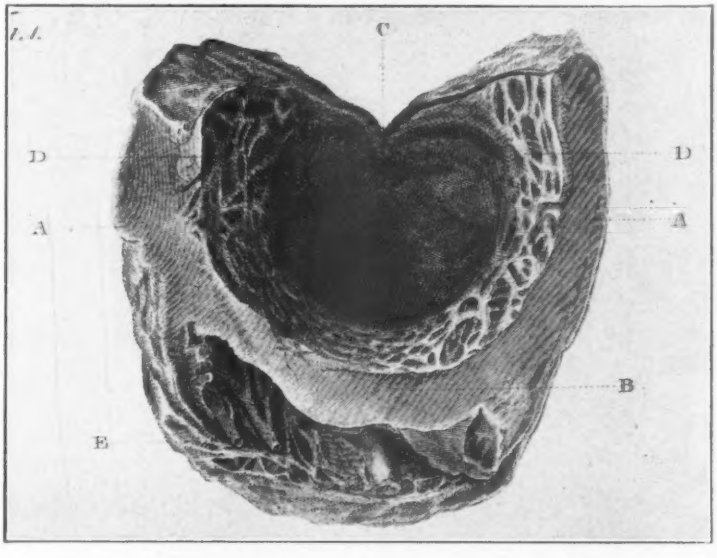

Fig. 1.—Represents an oblique section through the ventricles of the heart, where a part of the left ventricle near the apex was dilated into an aneurysmal sac. *A,A.* The fleshy parietes of the left ventricle. *B.* A part of the septum. *C.* The cavity of the aneurysmal bag which is lined with a thick smooth membrane. *D,D.* A part of the natural fasciculated structure upon the inside of the left ventricle. *E.* A small part of the fasciculated structure of the right ventricle, near the apex of the heart.*

eventually becomes the seat of an aneurysmal dilatation, as in Baillie's case, on occlusion of the descendens branch of the left coronary artery.

The following examples of aneurysm of the left ventricle have occurred in the Wisconsin General Hospital in the past year:

CASE 1.—A. K., white female, aged sixty-two years, housewife by occupation, was admitted to the Wisconsin General Hospital for the first time on July 25,

*In the preparation, from which this Figure was taken, the section has been made nearer to the diseased part than could have been wished. It would have given a more distinct idea of the disease, if more of the natural structure of the heart had been preserved. The preparation, however, was made many years ago.—From Dr. Hunter's Museum.

(From "A Series of Engravings, accompanied with Explanations, which are intended to illustrate the Morbid Anatomy of some of the most important Parts of the Human Body," Matthew Baillie, 1799 [1803].)

1926, complaining of weakness and shortness of breath. She gave a history of nausea appearing in May, 1926, succeeded by the vomiting of the food eaten immediately preceding. Three hours later the vomiting recurred, and at this time the vomitus was chocolate colored. Material of the same nature was later evacuated by bowel. On occasions since this original disturbance, there has been blood in the stool. Furthermore, shortness of breath on exertion developed, and loss of weight attended loss of appetite. Three weeks prior to admission there was observed some transient jaundice. In the past medical history there was determined a susceptibility to respiratory infections and the recurrence of influenza.

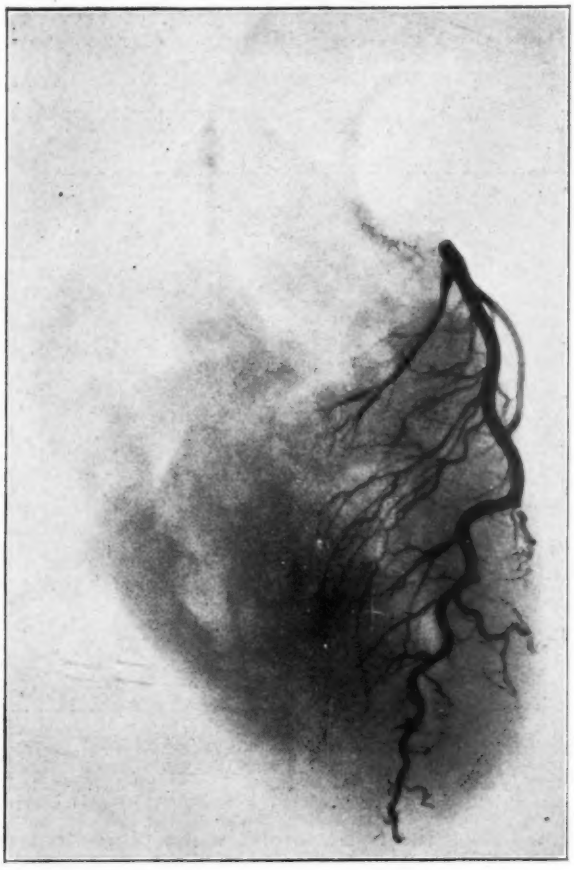

Fig. 2.—Lipiodol injection of the descendens branch of the left coronary artery.

Physical examination determined the important findings of evidences of fluid accumulation in the left thoracic base, distant heart sounds, blood pressure 120/75, tenderness in the left lower quadrant and under the right costal margin, râles and a pleural click at the left base above the level of fluid. An exploratory puncture removed from the left pleural space straw-colored fluid, which rapidly coagulated.

X-ray studies were made of the gastrointestinal tract with negative results. X-rays of the chest confirmed the presence of fluid in the left base and determined considerable increase in the hilum lymph nodes. Electrocardiographic studies showed notching of the R_2 on descending limb, deep S-wave in Lead III, and P_1 notched. The P-R interval was 0.18 seconds and the QRS complex occupied 0.08 seconds. On two or three examinations the uncatheterized specimens of urine showed faint traces of albumin and many pus cells. Examination on admission showed

53 per cent hemoglobin, and the red blood cells numbered 5,090,000; white blood cells 20,200 with 71 per cent polymorphonuclear neutrophiles, 1 per cent eosinophiles, 20 per cent small lymphocytes and 8 per cent large mononuclears. Subsequent counts showed a decline in this leucocytosis to 11,150. Four examinations of the stool showed no occult blood, ova, or parasites. Five examinations of the sputum were negative for tubercle bacilli. The blood Wassermann was negative. The blood chemistry showed no deviation from normal. The pleural fluid showed no tubercle bacilli on direct stain, and guinea pig inoculation was likewise negative.

From the above history and findings, a diagnosis of myocardial degeneration with coronary sclerosis and a complicating serofibrinous pleurisy was made. A febrile course ranging from slightly subnormal to 100.6° F. prevailed throughout the period of two weeks' residence in hospital.

The patient maintained a relatively inactive existence at home and was free from symptoms for almost four months, when an attack of diarrhea occurred.

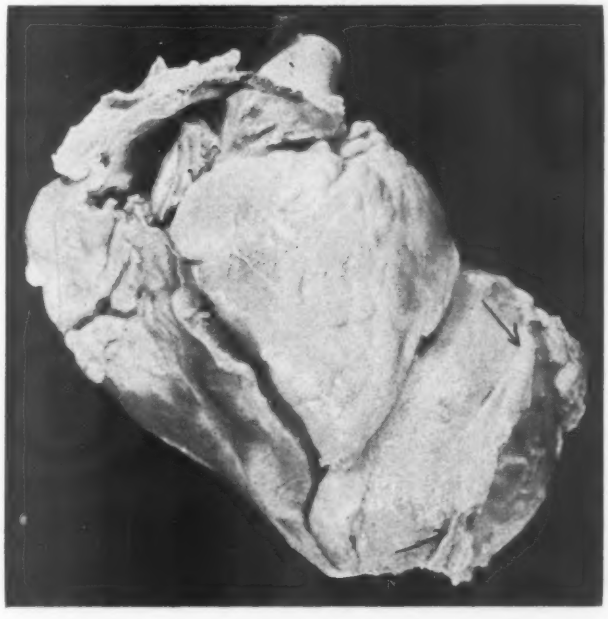

Fig. 3.—Heart from Case 1; arrows indicating adherent pericardium overlying the aneurysm.

This lasted for four days, the bowels moving from fifteen to twenty times a day. The patient was markedly debilitated by this time. She stated that during the period preceding the attack the stools had been orange-colored, but during the diarrhea they had become almost black. Dyspnea, restlessness, and productive cough recurred. The urine had been scanty since the onset of the diarrhea. On this readmission (December 1, 1926) there was determined a very slight jaundice, extreme orthopnea, oral sepsis, basal râles with definite fluid signs almost to the angle of the left scapula, cardiac enlargement—possibly somewhat obscured by fluid at the left base, indistinct heart sounds, blood pressure 134/85, tympanites, pretibial edema, suggestion of early cachexia, and a subcutaneous nodule at the site of the pleural puncture. An impression of a neoplasm with metastasis to the mediastinal structures and lung was gathered. The resistance in the epigastrium, more particularly to the left, was believed to represent either a primary source of such neoplasm or a chronic passively congested liver. The course from this time until the fatal termination, six weeks later, was marked by continuance of the

diarrhea with an increase in the epigastric mass, which was determined later to be quite definitely the liver, paroxysmal dyspnea, and occasional blood streaking of the rather scanty sputum. Two aspirations of 1000 and 150 c.c., respectively, were done. The nodule above described in the lower left chest wall was excised and proved to be inflammatory in nature. X-ray of the chest confirmed the fluid level displacing the enlarged heart to the right, and the left border of this organ was never capable of demonstration. The x-rays of the gastrointestinal tract showed only a possible colitis. Sputum examinations continued negative for tubercle bacilli. One urinalysis showed albumin with many hyaline and a few granular casts. Pus cells, persisted in these studies.

Against the opinion of a neoplasm should have been the maintained high red blood count, between 4,620,000 and 5,060;000, with the leucocyte count between 9,750 and 12,850. The hemoglobin ranged from 35 to 65 per cent. The stools

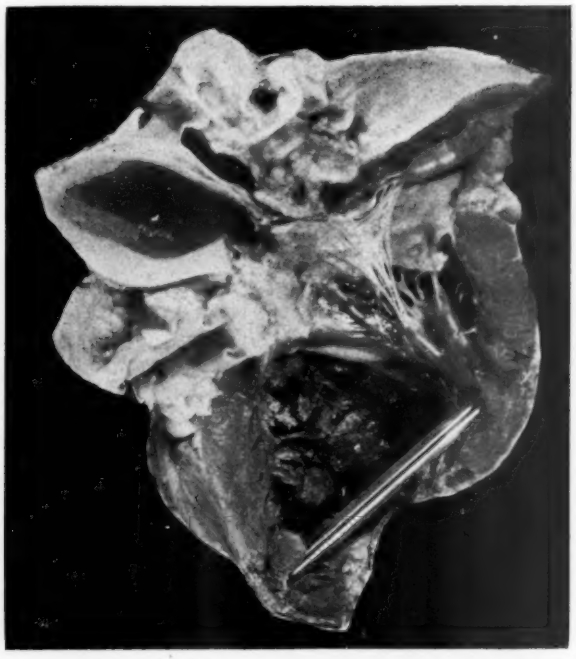

Fig. 4.—View of the interior of the left ventricle in Case 1. Note the thinness of the myocardium at the margin of the aneurysm and the contained mural thrombus within the sac.

showed some blood. The blood sugar was 132 mg. per 100 c.c., and the non-protein nitrogen, 57. The phenolsulphonephthalein output was 55 per cent in two hours.

The exitus of this patient was quite sudden and was characterized by marked tachypnea without change in pulse rate or volume.

A necropsy was performed two hours post-mortem with the following findings and conclusions:

Anatomical Diagnosis.—Hypertrophy and dilation of right heart, hypertrophy of left ventricle, aneurysmal dilatation of left ventricle due to atherosclerotic obliteration of the descending branch of the left coronary artery; old mitral endocarditis; extensive thrombosis of the pulmonary arterial branches; hemorrhagic infarct of the left lung; chronic passive congestion of the viscera; atherosclerotic nephritis; anasarca; localized pericardial adhesions over aneurysmal sac; advanced atherosclerosis of the aorta and its main branches; myomas of uterus.

Heart: Weight 655 gm. Pulmonic orifice, 6.5 cm.; tricuspid orifice, 12 cm.; mitral orifice, 9.5 cm.; aortic orifice, 7 cm.; left ventricle, 3 mm. to 2 cm.; right ventricle, 4 to 8 mm. The mitral valve shows fibrous thickening and is moderately stenosed. The right auricle and ventricle are markedly dilated. An aneurysmal dilatation of the left ventricle, adjacent to and somewhat involving the interventricular septum, begins 1.5 cm. from the tip of the ventricle. This aneurysm is filled with a laminated white thrombus, which is adherent to the ventricular walls, and measures 7 by 10 by 4 cm. Over its pericardial surface adhesions bind the two pericardial surfaces firmly together. The wall of the aneurysm appears to be composed entirely of dense gray fibrous tissue, and it varies from 2 to 3 mm. in thickness.

On examining the descending branch of the left coronary artery there is found a mural thrombus 5 mm. in length, 1 cm. from the beginning of the left coronary

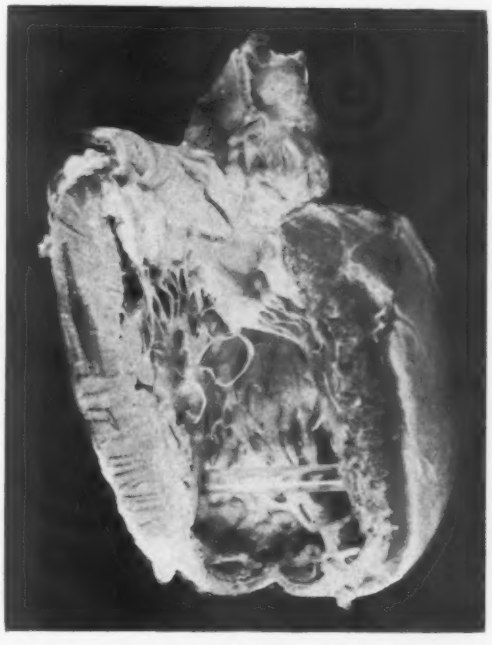

Fig. 5.—Heart from Case 2. Note thin wall of left ventricle in area of dilatation with organized mural thrombus beneath.

artery. Beyond the thrombus the artery is completely obliterated and appears as a firm, fibrous cord.

Microscopical examination of the heart shows a fibrous myocarditis with hypertrophy of the muscle cells. There is atherosclerotic obliteration of the descending branch of the left coronary artery.

Lungs: There is extensive thrombosis of practically every main branch of the pulmonary artery on either side. In the base of the left lower lobe there is a recent hemorrhagic infarct which measures 2 cm. in diameter. Aside from the above mentioned points, there is evidence of chronic passive congestion and moderate anthracosis.

Aorta: There is extensive atherosclerosis with many calcified and ulcerated plaques.

The findings in the remaining viscera are those to be expected in an individual of this age with cardiac decompensation. No evidence of malignancy is found.

The extensive pulmonary thrombosis with such a small amount of infarction of the pulmonary tissue would lead one to believe that the thrombosis had occurred shortly before death and was in all probability a very important factor in the death of the individual. The cardiac aneurysm would appear to have been of several months' duration. The obliteration of the coronary vessel had apparently been so slow in developing that there had been time for resorption of the dead

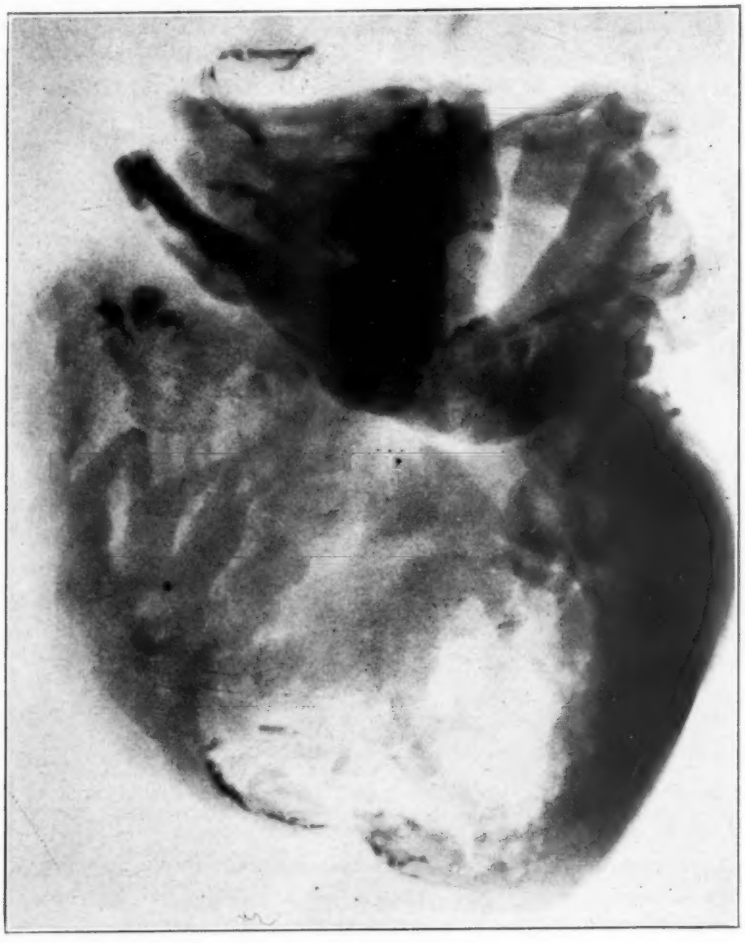

Fig. 6.—X-ray of excised heart from Case 2. Note calcification in the area of aneurysmal dilatation and in the coronary vessels.

cardiac muscle and its replacement by new connective tissue. The dense pericardial adhesions support this view. It would seem that such a condition would be incompatible with life unless its development were of a chronic nature.

Case 2.—G. McC., white male, eighty-five years of age, was first admitted to the Wisconsin General Hospital on December 11, 1925, with an attack of herpes zoster, which was complicated by post-herpetic neuritis. At this time extreme arteriosclerosis and emphysema with weak heart sounds alone were noted from a cardio-

vascular standpoint. A history, however, of an attack of hemiplegia in August, 1925, was significant. After a period of hospitalization of sixty-eight days, he was discharged on February 16, 1926, but returned eleven days later with the statement that four days previously, marked abdominal pains had occurred and edema of the feet and legs with shortness of breath had progressed from that time. Indeed, the night prior to admission he was unable to sleep because of orthopnea.

On physical examination total cardiac arrhythmia was discovered. The respirations were of the Cheyne-Stokes' variety and fine râles were heard in both bases. Marked edema existed in the feet and legs, and there was noted a positive centrifugal venous pulse in the radial veins. The response to venesection was prompt and maintained, the venous pressure falling from 21 to 18 cm. of water and the general condition becoming steadily better. Cyanosis which had been marked at onset, recurred from time to time, but digitalization in addition to the above blood-letting seemed adequate to maintain the patient in relative circulatory comfort. Gastrointestinal x-ray studies suggested a duodenal ulcer at this time. He was discharged on May 7, 1926, and maintained his relative equilibrium for several months, but late in October, 1926, he again became bed-ridden because of extreme weakness and dizziness. The occasion of his readmission was a fall from bed, leading to multiple contusions. From a circulatory standpoint the conditions were much as on the previous admission; auricular fibrillation with general arteriosclerosis, aortitis, and myocardial degeneration being the chief findings. Coffee ground vomitus was noted late in the course of his illness, and on January 14, 1927, he suddenly expired.

Necropsy, performed two hours post-mortem, revealed the following:

Anatomical Diagnosis.—Generalized atherosclerosis; aneurysm of left ventricle; atherosclerotic obliteration of descending branch of left coronary artery; diffuse and saccular aneurysms of the aorta; aneurysm of the splenic artery; softened mural thrombus in cardiac aneurysm; chronic duodenal ulcer; hemogastrium; atherosclerotic nephritis; old pulmonary tuberculosis; pulmonary emphysema; hypostatic pulmonary congestion; old pleural and peritoneal adhesions.

Heart: Weight 320 gm. Pulmonic orifice, 6.9 cm.; tricuspid orifice, 11.5 cm.; mitral orifice, 10.2 cm.; aortic orifice, 9.5 cm. There is present near the tip of the left ventricle a saccular dilatation, measuring 5 cm. in diameter. In this area the ventricular wall measures 3 mm. in thickness, is firm, gray, and shows areas of calcification. There is a softened mural thrombus attached to the endocardium in this region. The epicardium is gray and considerably thickened over this region. There are no pericardial adhesions. Above the area of dilatation the ventricular wall measures 1.8 cm. in thickness. Both coronary arteries show extensive calcified atherosclerotic plaques. The branch of the left coronary artery leading to the area of dilatation is completely occluded.

The heart otherwise shows nothing of especial interest.

Aorta: Throughout the entire extent there are numerous large atherosclerotic plaques, many of which are ulcerated. Some of the ulcerated plaques have thrombi covering the ulcerated areas. The organ is irregularly dilated throughout. At the level of the third lumbar vertebra there is a dissecting aneurysm 7 cm. in length which bulges anteriorly and is filled with a laminated clot. There is a similar but much smaller aneurysm of the splenic artery. There is but little calcification in any of the sclerotic plaques in the aorta.

Stomach: There is a blood clot of about one liter present in the organ. The mucosa shows no bleeding points. About 1 cm. beyond the pyloric sphincter is a chronic duodenal ulcer 2 cm. in diameter. A bleeding point is located in the crater of this ulcer. There is a considerable amount of clotted blood in the lumen of the small intestine.

Kidneys: Each kidney shows several retention cysts and numerous cortical scars. The remaining organs have no lesions pertinent to the present consideration.

Microscopical examination of the various organs confirmed the above anatomical diagnosis.

In this case it is apparent that the hemorrhage from the duodenal ulcer was the immediate cause of death. The cardiac aneurysm must have been of long duration, as evidenced by the calcified plaques in its wall. As in the preceding case, it was due to an atherosclerotic obliteration of the descending branch of the left coronary artery. Here again the process must have been one of long duration to allow resorption of the necrotic cardiac muscle and its replacement by scar tissue.

The two cases of aneurysm of the left ventricle herein recited presented widely variant clinical features. In Case 1 the occurrence of a gastrointestinal disturbance succeeded by symptoms and signs of decompensation had led to a conclusion of myocardial degeneration from coronary sclerosis on the first admission. Review of the subsequent course of events reveals no adequate reason for a revision of this opinion. Nevertheless, a suspicion, unconfirmed, of a new growth was entertained. The manner of termination in this case was very dramatic and was explained pathologically by the extensive pulmonary thrombosis. The constant presence of pleural fluid at the left base rendered remote or impossible a diagnosis of cardiac aneurysm by the x-ray, even though the deformity of the heart at necropsy was unmistakable. Subjective evidence of myocardial insufficiency coupled with the physical findings of decompensation in an arteriosclerotic, senile individual who later developed auricular fibrillation, offered little grounds for suspicion of cardiac aneurysm in Case 2. In neither case was the typical painful episode of coronary thrombosis experienced, although in Case 2 the patient had suffered indefinite abdominal pain prior to his circulatory collapse.

From the pathological standpoint, the etiological factor in both of the cases was the same; namely, coronary atherosclerosis leading to obliteration of the vessel and slow infarction of the heart muscle. The only difference in the cases is the extent of the infarction and the stage of repair at which the two lesions were seen. From these two cases it appears quite evident that the suddenness of the coronary occlusion, rather than the amount of damaged heart muscle, is the important factor in deciding the outcome in cases of cardiac disease due to coronary occlusion.

The ante-mortem recognition of cardiac aneurysm is unusual. However, a clearer conception of the clinical picture of coronary thrombosis and an appreciation of its sequelae should lead to a higher percentage of correct diagnoses of this condition in the future. Certainly it should be borne in mind and looked for whenever the acute phase of coronary thrombosis has been survived. According to Lutem-

bacher,[5] a localized point of pain and tenderness persists over the area of infarction. He further remarked the immobility of the heart on change of position by reason of the pericardial adhesions. Contrary to Smith[6] and other observers, Lutembacher maintained that no retraction of the parietes occurred over the point of pericardial adhesions on cardiac contraction, because of the altered contractile power of the underlying myocardium. Libman[7, 8] has repeatedly called attention to the presence of a pulsation more marked medial to the cardiac apex than at the apex proper, which, coupled with the dull first heart sound, has seemed significant in the diagnosis of cardiac aneurysm. Gallop rhythm, especially in the recumbent position, has also been remarked in these cases by Libman. At times the shape of the heart as revealed by the x-ray and its irregular functioning under the fluoroscope may be diagnostic.[6] Aside from these occasional aids in the diagnosis of cardiac aneurysm, the progressive development of decompensation and of arrhythmias succeeding coronary thrombosis are inferred evidences of aneurysm, but simply reflect the myocardial insufficiency attendant upon the coronary occlusion rather than an aneurysmal dilatation.

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SYSTOLIC BLOOD PRESSURE IN CARDIAC DECOMPENSATION AND DURING COMPENSATION*

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CHICAGO, ILL.

IT IS generally assumed that the systolic blood pressure falls in cardiac decompensation and that it rises as compensation becomes established. However, it has been observed that patients with marked cardiac failure and severe cyanosis may show hypertension. Sahli[1] first called attention to this increase in blood pressure and spoke of this phenomenon as "Hochdruckstauung." He observed this to be true not only for valvular lesions, but for cardiac failure due to emphysema and arteriosclerosis, explaining this paradoxical increase as being due to stimulation of the vasomotor center. Geisbock[2] confirmed these observations, but noted further that the blood pressure fell as the edema disappeared. Lang and Manswetowa[3] found an increase in blood pressure to be the rule in all mitral lesions and also in emphysema with decompensation, but reported it as occurring only in a small proportion of patients with aortic lesions. Durig and also Freshe[4] have disputed these observations. The latter in a study of seven hundred cases of heart disease could only find five with increase in blood pressure which he could attribute to decompensation. Loschkarewa[5] studied sixty patients with cardiac decompensation and observed a definite increase during decompensation, and a fall in systolic pressure as compensation became established.

We have studied thirty-five patients with severe cardiac decompensation. All showed signs, such as edema, dyspnea, and varying degrees of cyanosis. We did not include severe cases of auricular fibrillation in this group. Blood pressure readings (mercury manometers) were taken on the day of admission and thereafter at varying intervals of from three to five days and on date of discharge. The treatment received was rest in bed, digitalis, and opium when indicated. Three patients were used as controls and received no medication.

RESULTS OF PRESENT STUDY

In sixteen (45.7 per cent) of thirty-five unselected cases with severe cardiac decompensation, the systolic blood pressure fell from 10 to 40 mm. as compensation became established. Of these, fifteen were improved and discharged; death occurred in only one patient in whom the blood pressure fell.

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In twelve patients (34.2 per cent) the systolic blood pressure remained unchanged. Of these, nine were improved and discharged and three died.

In five patients, or 14.2 per cent, the blood pressure was increased as compensation occurred, and of these, three died and the remaining two were improved.

In two instances there was a definite fall in blood pressure as compensation occurred, but the blood pressure rose to that on admission before the patient left the hospital.

TABLE I

NUMBER OF CASES SHOWING CHANGE IN BLOOD PRESSURE

	FALL	RISE	NO CHANGE	FALL THEN RISE
Aortic Regurgitation Aortitis, Decompensation	5	4	4	2
Mitral Regurgitation	3		1	
Mitral Stenosis			2	
Chronic Nephritis with Cardiac Hypertrophy and Decompensation	2			
Emphysema, Chronic Myocarditis, Decompensation	3		3	
Chronic Nephritis	3	1	2	

In fifteen patients with aortic regurgitation, there was a definite fall in the systolic blood pressure with return of compensation in five. The fall in blood pressure varied from 10 to 40 mm. These patients were all improved and discharged from the hospital. In four patients a rise in blood pressure of from 10 to 25 mm. occurred, and was present at the termination of the cases. Three of these died; the fourth was discharged as improved. In four other patients there was no change in blood pressure, and of these only one died, the others being discharged as improved. In two patients the blood pressure fell from 10 to 25 mm., and then rose to that on admission, although the patients were clinically improved.

In six patients with chronic myocarditis, emphysema, and cardiac decompensation, the blood pressure fell from 20 to 40 mm. in two instances with improvement; in one patient, a fall of 14 mm., with death; in three, no change in blood pressure, with death in two and in the final instance the patient was improved.

Among four patients with mitral regurgitation, a fall in blood pressure occurred in three, and in one there was no change. All of these were improved. In two patients with mitral stenosis, there was no change in blood pressure. Among six patients with chronic nephritis, a fall occurred in three patients, a rise in one patient, and no change in two. All of these were discharged as improved.

DISCUSSION

Our observations direct attention to the fall in blood pressure which occurs as cardiac compensation is established. The increase of systolic blood pressure at the time of decompensation was necessarily inferred because of the fall in pressure and the maintenance thereof as the patient improved. Among eight patients readmitted for recurring decompensation, we were able to note in five that the systolic blood pressure was from 20 to 40 mm. higher on the second and third admissions than on the first and second discharges. The explanation of an increase in blood pressure appears paradoxical when this is viewed as a mechanical or dynamic problem. Reflex or increased tonus of the vasomoter center, as suggested by Sahli, offers an explanation. It is of interest to note that Cobet[6] has studied experimentally the relation of dyspnea and cyanosis to blood pressure, and finds the changes in blood pressure proportional to the CO_2 content of the blood. Cyanosis, however, is not a frequent occurrence in aortic lesions, and it is

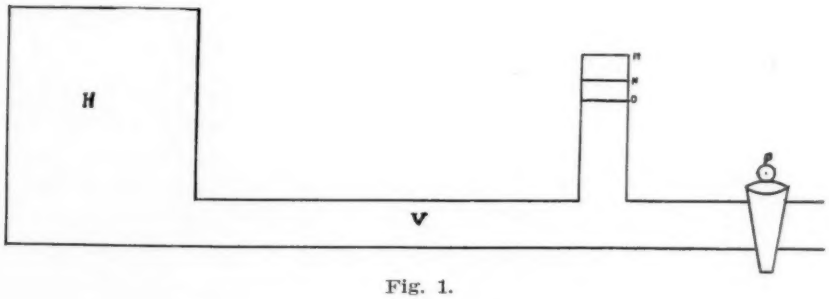

Fig. 1.

difficult to explain the rise in blood pressure on this basis. Lang and Manswetowa[3] offer the accompanying diagram (Fig. 1) as a possible mechanical explanation:

In Fig. 1 let *H* represent the heart, and *V*, the arterial system. Under normal conditions, with no increased resistance, the blood pressure is represented on the manometer as *O*. Assuming an increase in peripheral resistance by turning *P*, the blood pressure will rise to point *M*. This increase in peripheral resistance in cardiac decompensation is a stasis in the arterial system. Allowing then for this quantity of blood from *H*, the blood pressure will rise to *N*, which is still above the normal blood pressure.

The possible rôle of passive congestion of the kidney, as a determining factor in the production of the hypertension, is also to be considered. Our studies have failed to prove this definitely, for the patients who did not show any changes in blood pressure readings showed the same evidence of passive congestion of the kidney (as evidenced by urinary changes) as those that did manifest a rise and subsequent fall.

Geisbock[2] inferred that, in decompensation with edema, a compression of the smaller arteries and capillaries occurred which produced an increased peripheral resistance and thus explained the increased pressure and also the fall as the edema disappeared. We were able to observe the fall in pressure with the general improvement of patients, but we do not accept Geisbock's view, since it is well known that generalized edema may occur in many conditions without changes in blood pressure.

SUMMARY

The systolic blood pressure fell in 45.7 per cent of unselected cases of cardiac failure as compensation became established. We observed this fall in cases of aortic regurgitation, mitral regurgitation, chronic myocarditis, emphysema, and chronic nephritis.

It is probable that a combination of mechanical factors and increased tonus of the vasomotor center are responsible for the initial rise in systolic blood pressure during decompensation.

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PULSE RATE STUDIES WITH THE PULSE RESONATOR*

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IN 1926, Goldschmidt, Kraus, and Seelig[1] described a new method of recording the human arterial pulse by means of a very ingenious apparatus which records changes of rate directly on a moving paper, called the "pulse resonator" by its inventor, Goldschmidt. The pulse resonator utilizes the phenomenon of resonance to register the variations of the pulse rate. The mechanical impact of the wall of the pulsating radial artery is used to make and break rhythmically the circuit of an electromagnet. The resultant magnetic field acts upon a series of twenty-four pendula of different lengths. Each pendulum is tuned to a different period and will start swinging only in resonance with a certain given rate. Change of rate will activate another pendulum. The movement of every swinging pendulum appears as a horizontal line on the revolving paper roll of a connected recording device. The oscillations of the individual pendulum are so damped that only pendula representing lower frequencies than the one which corresponds to the true pulse rate will be set in motion, never the higher ones. Therefore, the highest recorded frequency represents the actual rate at every point in the tracing. This is a very important point to be considered in interpreting the pulse resonator tracings. Such a tracing (Fig. 1) shows a number of horizontal parallel lines. The individual lines are interrupted at intervals of varying lengths. According to the physical properties of the instrument, at every point in the tracing the uppermost line represents the actual rate. The apparatus records rates between 30 and 192 per minute.

This pulse tracing gives a comprehensive graphic picture of the fluent changes in rate. It indicates the rate directly at every point of the tracing. The accuracy of the pulse resonator was checked by the inventor by means of a device which produced artificial pulsations which could be regulated in rate and rhythm, and absolute fidelity in recording the changing rate is claimed. In analyzing the pulse resonator tracings, however, we deemed it indispensable to correlate the findings with those obtained by a method registering every individual pulse or heartbeat, and permitting the calculation of the true rate from beat to beat. We found no reference to such a control in the original publication of Goldschmidt, Kraus, and Seelig.

Electrocardiograms and pulse resonator tracings were taken simultaneously on the same individuals. The comparative analysis of the

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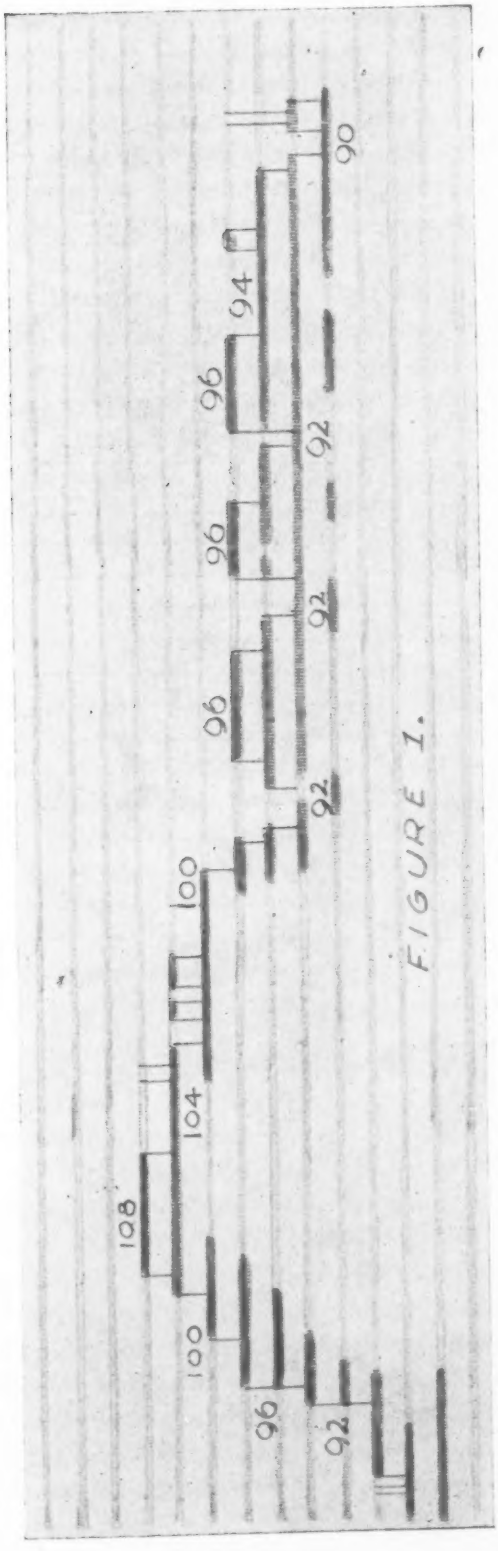

Fig. 1.—Pulse resonator tracing.

two tracings was laborious, since the speeds of the film roll of the electrocardiogram and the paper roll of the pulse resonator differ greatly. Records covering several minutes were taken, the synchronous start and ending of the two tracings carefully checked with a stop watch, and every minute marked on both records. Then the intervals between every two ventricular complexes of the electrocardiogram were measured and the rate for every beat calculated. The figures obtained were plotted on a curve, the abscissa of which was chosen to be of the same length as the actual length of the corresponding pulse resonator tracing; usually one minute represented 15 centimeters. Every point on the curve so constructed corresponded in time exactly with the pulse resonator tracing, and could therefore be superimposed upon it and compared with it.

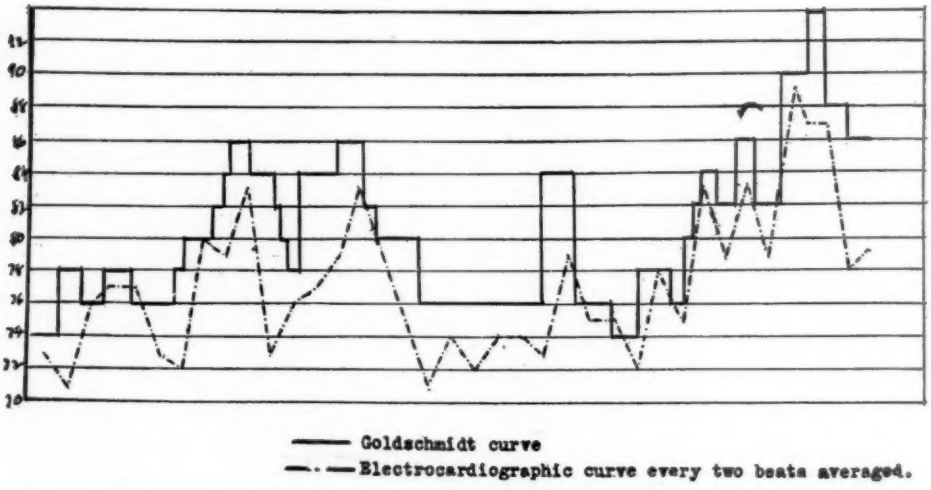

Fig. 2.—Simultaneous pulse resonator and electrocardiographic curves of heart rate.

Comparative study of the two tracings shows a correspondence in general outline rather than in detail. This becomes more apparent when the pulse resonator curve is compared with a tracing obtained by plotting the averages of the intervals between three or four ventricular complexes. Fig. 2 shows a close resemblance between the two tracings, one of the closest in our series; but even here there are marked discrepancies. The pulse resonator curve registers higher frequencies practically throughout the entire period. Differences in rate amounting to 8 or 9 per cent can be found at two points. In addition, the pulse resonator tracing fails to follow the sudden and short changes in frequency registered by the electrocardiogram. The general trend of the tracings is identical, however, as appears especially well by comparing the pulse resonator tracing with the curve obtained by plotting the average of two successive pulse intervals (Fig. 3). The inadequacy of the pulse resonator in following sudden

short drops of frequency is again demonstrated in Fig. 4. The contrast is more pronounced since the changes of frequency are more frequent and sudden, as well as more extensive than in the former case. The correspondence in general tendency is shown again in the "average" curve (Fig. 5). Again the curve computed from the electrocardiogram of a patient with sinus tachycardia shows variations of rate between 145 and 150, and only a short period during which the rate is fixed at 150; whereas, the pulse resonator registers an uninterrupted line representing a rate of 152 (Figs. 6 and 7).

It is our impression that the pulse resonator tends to register rates higher than the electrocardiogram, which, after all, records the true rate. It will be recalled that the uppermost line of the pulse resonator tracing must always be considered as representative of the true rate. Very often it is impossible to decide whether or not an indication of

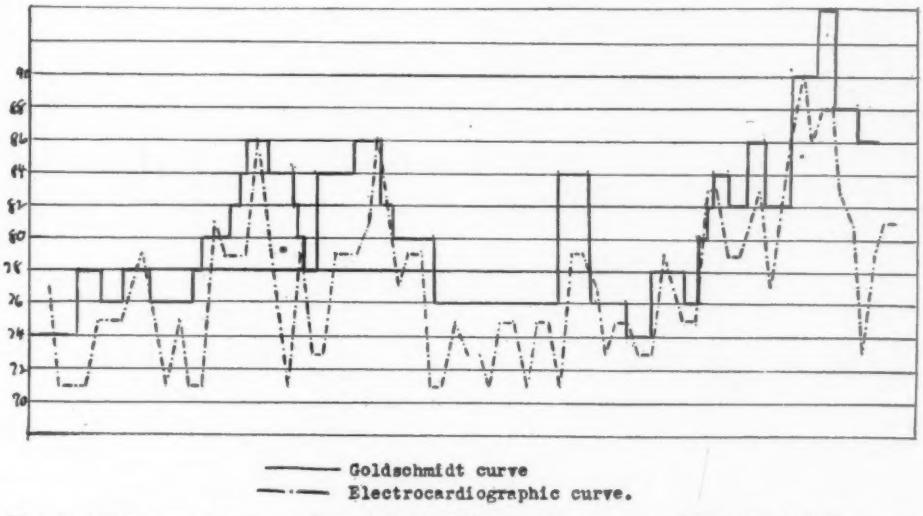

Fig. 3.—The same curves. The electrocardiographic curve was constructed by averaging every two beats.

a line, short in duration and faint in appearance, should be accepted as significant. This uncertainty in interpretation may account, in part, for the high rates. It is probable, however, that there are other factors inherent in the physical properties of the apparatus that are responsible for the lack of correspondence between the pulse resonator and electrocardiographic tracings.

Although the pulse resonator may not always record the exact pulse rate, there is no doubt that it indicates its general trend and the range and periodicity of its changes. The record can be taken over a long period of time, while the person who is being studied can execute various test movements. Probably the greatest advantage of the pulse resonator is that it makes calculating and plotting unnecessary—a factor of real value in the study of long records.

While studies of the changes of normal sinus rhythm under different physiological conditions are numerous, modern investigations of the changes of rate over prolonged periods of time are very scant. Curves similar to ours were plotted by Mosler[2] from pulse and electrocardiographic tracings, recording up to 110 consecutive beats. He found differences of from 0.008 to 0.109 seconds between two consecutive pulse intervals with the patient at rest, and from 0.035 to 0.317 seconds after exertion. After exercise among 371 consecutive pulse intervals that were measured, two successive intervals were equal in 9.4 per cent, three successive intervals were equal in 1.3 per cent, and in no instance were four successive beats equal. No periodicity characterized these variations, nor was there any constant relationship

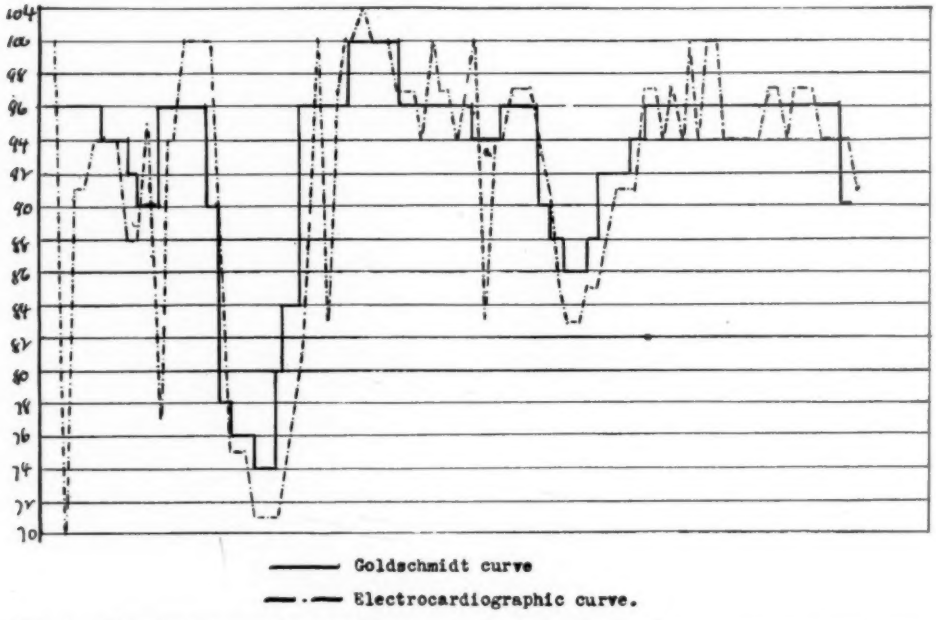

Fig. 4.—Simultaneous pulse resonator and electrocardiographic curves of heart rate.

to the rhythm of respiration. An excellent review of the older literature is given by Tigerstedt.[3] Aulo,[4] employing a special sphygmograph, registered the radial pulse over a period of three hours. He found differences in rate between beats as high as 10 per minute. His curves, too, resemble our curves that were calculated from the electrocardiogram.

Development of new methods of observation is always followed by new problems; and so in the case of the pulse resonator, Goldschmidt, Kraus, and Seelig point out in their first article a number of new psychological, physiological, and pharmacological problems that can be studied with the new instrument. They lay great emphasis on the differentiation of pulse, showing unduly wide ranges of variability of rate from those that are almost fixed. They find in most

cases a more or less periodic alternation of the frequency of the pulse, with a variation of about 8 per cent, quite independent of respiration. They call this a "double rhythm," and build a very complicated theory of the control of pulse frequency on this observation. It is generally known that variations of sinus rhythm occur independent of the phases of respiration. The theory of the "double rhythm" of the heart needs much more experimental support before it can be accepted. In their interpretation of their records they speak of the analyzed heart rate, by which they undoubtedly mean a rate determined by a harmonic analysis of the Fourier type. The physical significance of this analyzed heart rate is not clear. Furthermore, the concept of the analyzed heart rate cannot be used interchangeably

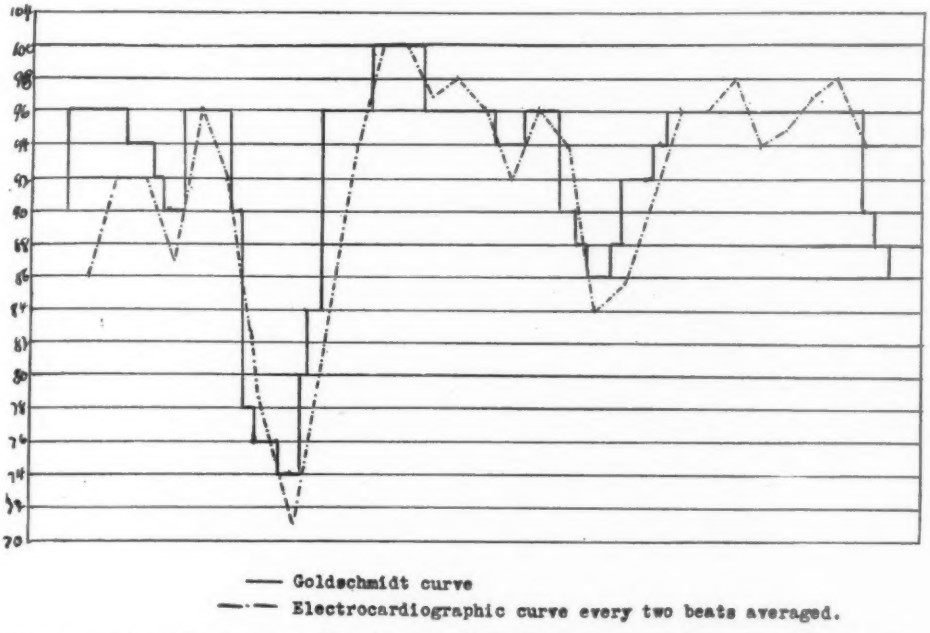

Fig. 5.—The same curves. The electrocardiographic curve was constructed by averaging every two beats.

with the usual concept of the heart rate, namely, as measured by the time interval between successive beats.

After the completion of our studies there came to our notice a critical analysis of the pulse resonator by Goldscheider.[5] He points out that the instrument records group arrhythmias rather than those of individual pulse beats, for as Mosier showed fifteen years ago, two successive pulse intervals are rarely equal. Goldscheider further states that Goldschmidt claims now to have made the apparatus so sensitive that the pendula will respond to variations from beat to beat, and rightly points out that this would invalidate the theoretical basis of the pulse resonator; for such a response could not be determined by periodic changes in the magnetic field. He discounts Kraus'

theory of the "double rhythm," and points out that the phenomenon depends, rather, on the natural variability of the heart beat due to vagus and sympathetic influences.

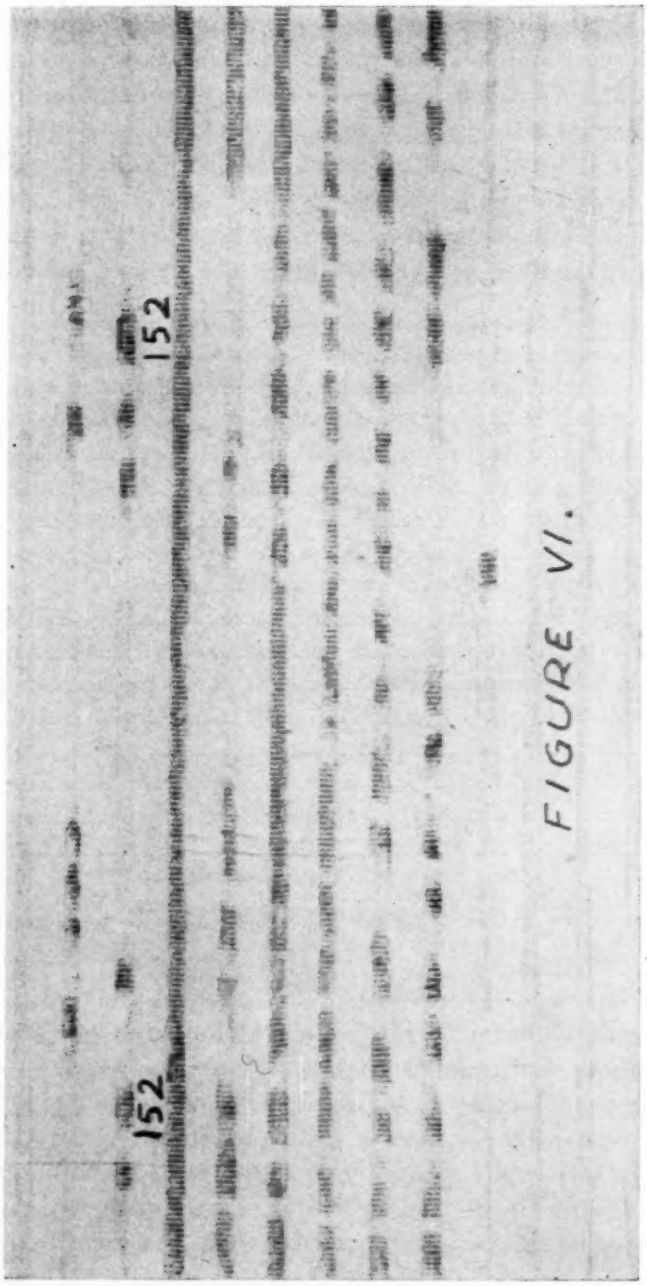

Fig. 6.—Pulse resonator trading of a sinus tachycardia.

The pulse resonator does not mirror accurately the quick and brief alteration in rate on which the original authors place so much emphasis. It is our conviction that the value of the instrument lies rather in the possibility it offers of taking long records without the

necessity of laborious calculations. Thus, it facilitates the study of such phenomena as occur periodically or aperiodically at long intervals, provided that the phenomena in question extend over at least four or five heartbeats.

SUMMARY

Tracings made with Goldschmidt's pulse resonator, an apparatus that records the constantly changing pulse rate over long periods of time, were checked with simultaneous electrocardiographic records. It was found that the new apparatus does not react quickly to sudden alterations in rate of short duration, and that it may indicate rates not accounted for in the electrocardiographic tracing. It does, however, record the general trend of the pulse rate with considerable fidelity. The pulse resonator should be useful in the prolonged observation of the pulse rate, and especially of phenomena which extend over more than four or five heartbeats, and which do not demand strict quantitative accuracy.

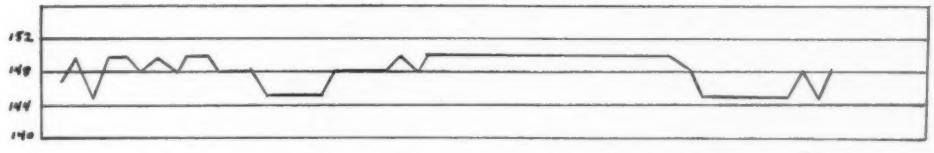

Fig. 7.—Simultaneous electrocardiographic curve of heart rate.

We wish to express our appreciation of the very valuable help rendered by Miss Jeanne Seeley in calculating and plotting the curves, and to Dr. Goldschmidt for his courtesy in placing a pulse resonator at our disposal.

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Department of Clinical Reports

PAROXYSMAL TACHYCARDIA WITH AN UNUSUALLY RAPID HEART RATE IN A BOY OF FOURTEEN YEARS

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CARDIAC arrhythmias occurring in children have been reported with increasing frequency during the last few years, particularly since the advent of the electrocardiograph.

The case herewith presented reveals an irritation of the pacemaker in which the ventricular rate is very rapid and responds to each auricular impulse.

A boy, aged fourteen years, applied to the school doctor for a permit to work and was referred to the Consulting Heart Board of the city schools, because of a very rapid pulse. He came before the Board for examination four days later, at which time he stated that he had had no serious illnesses and denied a history of rheumatism or sore throat. He further stated that he felt perfectly well when first examined.

Examination.—Weight was 120 pounds, and height 5 feet. Patient was well nourished, and was not acutely ill. He showed, however, rapid visible pulsation in the neck, but no palpable thrills. The apex beat was visible and palpable inside the nipple line in the fifth interspace. Apex rate was 160; radial pulse rate 160, with no irregularity of the pulse. Blood pressure 160/70 mm. An electrocardiographic tracing was made on the same day several hours later (Fig. 1). According to the technician, the boy experienced no uncomfortable sensations during the taking of the record.

The patient was again seen six days later. At this time the apex rate was 160 and regular. Two hours later, following examination, electrocardiographic tracings were taken which showed a ventricular rate of approximately 90 and a marked sinus arrhythmia (Fig. 2). The sinus arrhythmia disappeared promptly on exercise, as shown by Lead II of the succeeding electrocardiogram (Fig. 3). Roentgenographic pictures taken at two meters suggest the possibility of hypertrophy of the left auricle (Fig. 4). This is further demonstrated by fluoroscopy in the oblique positions which show the auricular shadow encroaching upon the posterior cardiac clear space.

A period of rest was advised.

The patient was seen again on September 27, 1927. He stated that he had been working hard all summer and that he had experienced no symptoms of distress. Physical examination revealed a pulse rate of 90 which increased to 150 after moderate exercise (hopping 25 times on each foot). The pulse rate returned to its previous level after two minutes. On two later occasions the patient has been seen, when the heart rate has been 90.

COMMENT

Lead II is particularly striking, revealing a ventricular rate of approximately 300. There is some evidence of right axis deviation both before and after the tachycardia has ceased. Differentiation must be made in this case between supranodal paroxysmal tachycardia and auricular flutter. The fact that the heart rate decreased gradually

and was found to be 160 at the apex is suggestive of auricular flutter. On the other hand, the absence of any signs of congestive heart failure is a point in favor of paroxysmal tachycardia. A normal rhythm and pulse rate was observed between attacks, and this would point to the diagnosis of a simple paroxysmal tachycardia of auricular origin, with a very rapid rate. It is probable that when the rate was observed at 160, there was present a 2:1 block. The very marked sinus arrhythmia, amounting almost to a so-called sino-auricular block, may be in

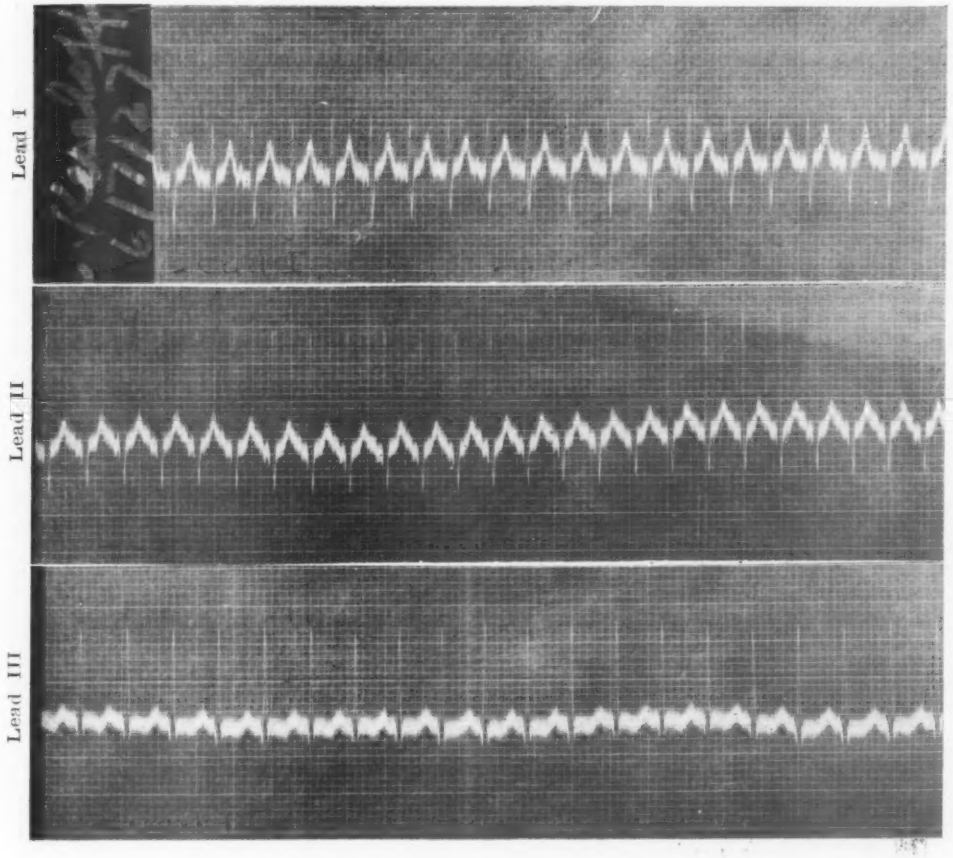

Fig. 1.—Auricular and ventricular rate 300 per minute.

this case added evidence of auricular damage. The P-waves tend to be notched in Lead I and are rather flattened in Lead II (Fig. 2). Again one sees a variation in the shape of the P-waves before and after exercise, as shown in Lead II (Figs. 2 and 3).

Auricular paroxysmal tachycardia would seem to be a probable diagnosis in this case since a 1:1 A-V ratio was observed.

Auricular flutter usually occurs with the ventricle responding to every second, third, or fourth auricular impulse and is usually associated with some symptoms of congestive heart failure. Willius[1] reports a very interesting occurrence of auricular flutter associated with

complete heart-block. Poynton and Wyllie[2] report a case of 1:1 flutter in infancy. Their patient was observed first when four months of age, with symptoms of acute congestive heart failure. At three and one-half years of age, the heart rate was consistently around 150 beats per minute.

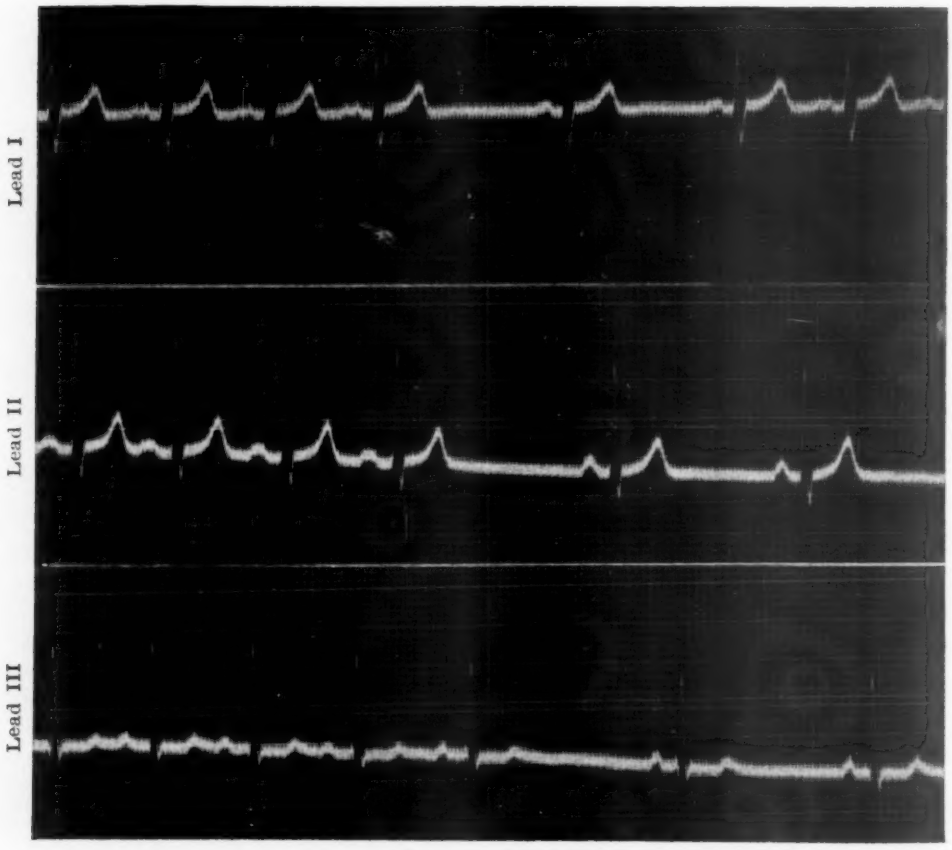

Fig. 2.—Sinus pauses. Rate 90 per minute.

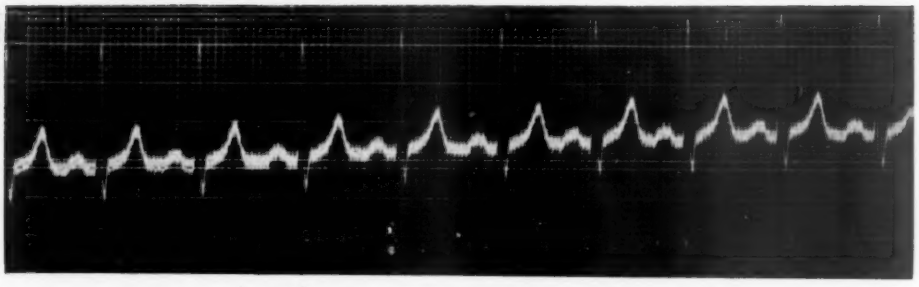

Fig. 3.—Lead II after exercise.

The arguments against auricular flutter in this case are: first, a 1:1 A-V ratio in flutter is very unusual; second, with a ventricular rate of 300, signs of heart failure would most certainly be present if the case were one of flutter.

Koplik[3] reported three cases of paroxysmal tachycardia occurring in childhood with ventricular rates respectively of two hundred and fifty, two hundred and seventy, and two hundred and eighty-eight beats per minute.

The differential diagnosis between auricular flutter and auricular paroxysmal tachycardia may sometimes become quite difficult, as in this case. In tachycardia one rarely finds the rate over two hundred and forty or two hundred and sixty per minute. In view of the fact that most of our records have been taken from the cases of adults, it

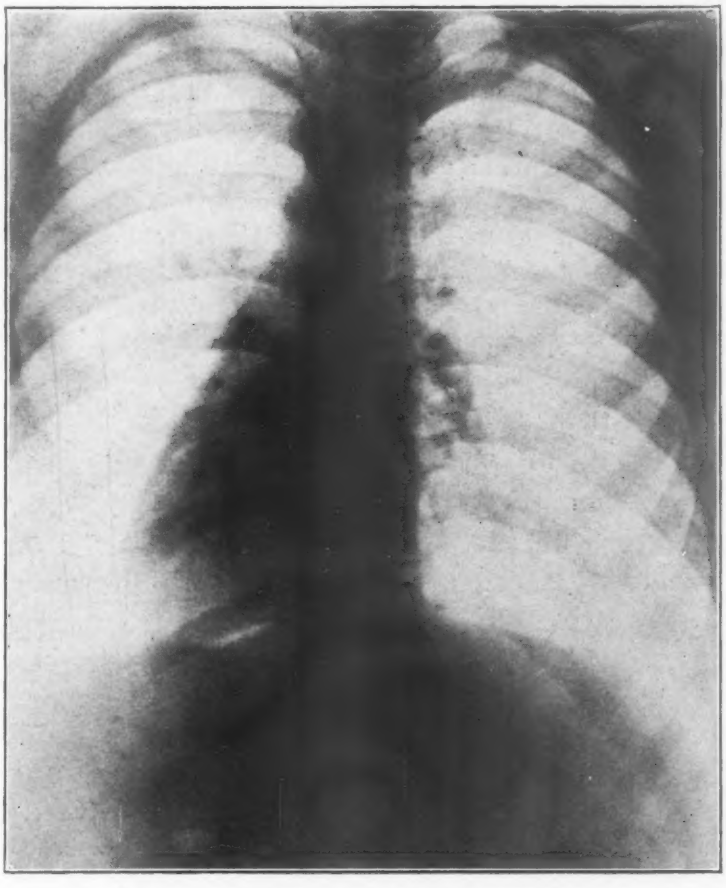

Fig. 4.—X-ray of the heart at 2 meters.

is quite possible that children may have a much higher ventricular rate, as reported in Koplik's cases and as shown in the above case.

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Department of Reviews and Abstracts

Selected Abstracts

Hay, John, Jones, H. Wallace, and Ince, Phoebe: The Action of Digitalis in Cardiac Failure With Normal Rhythm. Quart. Jour. Med., 1927, xxi, 153.

The authors have studied the effect of digitalis medication in a group of nine patients admitted to the hospital with congestive heart failure and normal rhythm. After a preliminary period of observation and standardization, digitalis was administered by mouth until the onset of nausea and vomiting or other toxic symptoms.

The authors observed a definite fall in heart rate with remarkable frequency occurring in 7 of the 9 cases. Only two of the patients had definite edema. There was a loss of weight during the observation period so that they assume that digitalis causes diuresis in those patients with edema. There was no alteration in blood pressure either systolic or diastolic. In the majority of cases the usual changes in the electrocardiogram, due to the influence of digitalis were well marked. Vital capacity was increased during the treatment in six cases.

Clarke, R. D., Coombs, C. F., Hadfield, G., and Todd, A. T.: On Certain Abnormalities, Congenital and Acquired, of the Pulmonary Artery. Quart. Jour. Med., 1927, xxi, 51.

Five cases of primary disease of the pulmonary artery are reported. In two the lesion was congenital. In the third and fourth an arteriosclerotic change was found without obvious cause. In the fifth, the lesion was syphilitic. Arguments in support of the view that there is an inherited factor in the causation of such lesions as were found in the third and fourth cases are brought forward. The nature of this inherited influence at work in these patients is not apparent from a study of the cases.

Parkinson, John and Bedford, D. Evan: The Course and Treatment of Auricular Flutter. Quart. Jour. Med., 1927, xxi, 21.

The authors report observations made on 52 patients with flutter that have been under care between the years of 1913 and 1926. From their study they conclude that flutter is usually an established rhythm when first diagnosed and is unlikely to cease or to change to fibrillation spontaneously.

Flutter most commonly occurs in middle age or later. In this series, males were affected about ten times as frequently as females. The condition is more often associated with chronic rheumatic heart disease or with cardiac enlargement in elderly males.

The results of treatment by digitalis, strophanthin and quinidine are presented in detail. They believe that digitalis removes flutter far more often than does quinidine. Digitalis alone restores normal rhythm in over one-third of the cases and in another third it induces fibrillation. Quinidine converts flutter directly to normal rhythm in about one case in five. It often cuts short paroxysms of flutter, though continued small doses do not entirely prevent their recurrence. Where digitalis has left fibrillation, quinidine may convert this to normal rhythm. The indications for digitalis and quinidine respectively are discussed.

The prognosis in this condition depends on the underlying heart lesion and on the reaction of the superimposed flutter to treatment. When the onset or flutter rapidly induces congestive heart failure, the expectation of life is far less than in the absence of failure.

Stone, C. T., Vanzant, Frances R.: Heart Disease as Seen in a Southern Clinic. Jour. Am. Med. Assn., 1927, lxxxix, 1473.

The material from which this study was made comprised all the definite cases of structural heart disease admitted to all services of the hospital in Galveston, Texas, from Jan. 1, 1920, to Dec. 31, 1926, inclusive. During this time the total number of hospital admissions was 25,816. Of this number 17,670 were white persons and 8,146 were negroes, a ratio of whites to negroes of 2.2 to 1. Of these patients 915 presented definite evidences of heart disease, and of these 501 were white and 414 were negroes, which shows that heart disease is 1.8 times more common in the negro race than in the white. Only the structural diseases of the heart were considered. Two hundred and ten patients came to autopsy.

Of the 915 patients with heart disease, 47.7 per cent belonged to the group classified etiologically as hypertensive; 19.3 per cent were syphilitic, 13.7 per cent were arteriosclerotic, 7.3 per cent were rheumatic, 2.3 per cent had angina pectoris, 1.5 per cent showed subacute bacterial endocarditis, 1.3 per cent were thyrotoxic, 0.7 per cent had congenital heart disease, 4.9 per cent were impossible of classification, and 1.3 per cent were grouped under the heading of "miscellaneous."

Further study on 436 patients with hypertensive heart disease showed that 274 were males and 162 were females. There was no striking difference in this series between the number of whites and negroes, but since the incidence of heart disease in the negro is practically double that in the white race, it would follow that hypertensive heart disease is twice as frequent among the negroes.

Of the 177 cases of syphilitic heart disease 103 were negro males as against 41 in white males, a total of 144 males to only 33 females. Syphilitic disease is therefore, about 4⅓ times more common in males than in females and in females it is about 6½ times more frequent in negroes than in whites. This is not due altogether to a higher incidence of syphilis among the negroes, because syphilis was found in this clinic in almost the same ratio in whites and negroes of the same social scale admitted to the hospital. Apparently then the cardiovascular apparatus of the negro is more susceptible to infection than that of his white brother. The negro males of this series were almost exclusively those who did very hard manual labor. It is this fact, plus syphilis, together, perhaps, with a lowered resistance which accounts for the high ratio of syphilitic heart disease among them. The low incidence of acute rheumatic fever in this series is of considerable interest. In the seven years during which the entire series of heart cases were admitted to the hospital, there were only 10 cases of acute rheumatic fever and 9 cases of chorea in the same clinic.

The observations in the 210 cases of the series which came to autopsy substantiates the accuracy of the classification according to the etiology of those cases seen only in the clinic. Tables show essential agreement with the clinical diagnoses.

The authors believe that heart disease as a whole is in the South a somewhat different problem from that seen in the East and North. The difference arises chiefly from two large factors: (1) the relatively low incidence of acute rheumatic fever and chorea in the South and (2) the proportionately larger negro population in the South.

Saphir, O., and Scott, R. W.: The Involvement of the Aortic Valve in Syphilitic Aortitis. Am. Jour. Path., 1927, iii, 527.

The authors have studied the necropsy findings of 71 cases in order to determine the alterations which occur in the aortic valve leaflets in relation to changes in the aorta. All cases showed fusion between the lateral portion of the leaflets of the aortic valve and the aortic valve at the sinus of Valsalva. This fusion between the lateral part of the cusps and the aorta apparently leads to a widening and a separation of the commissure. Sixty cases showed hyaline plaques of varying sizes in the region of the commissures. The central portion of the free margin of the cusps in a majority of cases showed a marked thickening.

The most extensive lesions of the aorta are found in a segment of about 4 cm. above the aortic valve. Intimal thickening, marked hyalinization, and sometimes calcification were present and in addition, varying numbers of depressed scars and longitudinal wrinkles. These areas

are supplied by vasa vasorum in large numbers. Since syphilis is a primary disease of the vasa vasorum, it appears that in a vast majority of cases there will be an involvement of this segment of the aorta.

Saphir, Otto, and Cooper, George W.: Acute Suppurative Aortitis Superimposed on Syphilitic Aortitis. Arch. Path. and Lab. Med., 1927, iv, 543.

The authors report a case of acute suppurative aortitis occurring in a patient with syphilitic aortitis. The lesions in the aorta were found in and about the aneurysmal dilatation, though there were also acute vegetative lesions on the aortic valve. Blood cultures showed diplococcus lanceolatus. The acute suppurative lesions of the aorta were formed exclusively in the intima and inner portion of the media which areas are supplied not by the vasa vasorum but by the circulating blood. The aorta showed typical syphilitic changes involving the coronary vessels and media.

Eyster, J. A. E., and Middleton, William S.: Venous Pressure as a Guide to Venesection in Congestive Heart Failure. Am. Jour. Med. Sc., 1927, clxxiv, 486.

The authors have studied twenty-one patients with cardiac decompensation that had been subject to venesection. Dyspnea, orthopnea, Cheyne-Stokes respirations, cyanosis, cough, bloody frothy expectoration, pulmonary congestion and edema, positive centrifugal venous pulse, engorged pulsating liver and venous hypertension have been the criteria for decision in the matter of blood letting. A venous pressure of 20 cm. of water, which was ascending, was the ultimate determining factor in the matter. As a rule 500 c.c. of blood were withdrawn.

Immediate improvement was marked in fifteen of the twenty-one patients, and no evident preexisting circumstances of age, sex, basic lesion or etiology was operative in this determination. The antecedent height of venous pressure was apparently without influence in this direction, and the degree of venous pressure fall bore no constant relationship to the early improvement. In six cases there was no apparent improvement from the venesection. Of these six cases, death occurred within forty-eight hours in four, and in twenty and ten days respectively in the remaining two. The ultimate outcome of the other cases was very unfavorable, 15 patients of the group dying in the hospital.

The authors believe that the immediate effect of venesection is to lessen the load of returning blood to the right heart. The effect is essentially mechanical. The authors point out and emphasize that venous pressure is an excellent index of the myocardial reserve, and venous hypertension, in the absence of local causes, results only from

cardiac decompensation. Routine venous pressure determinations are, therefore, advised, not only as a guide in venesection, but also as a result of its efficacy.

Griffith, J. P. Crozer: Displacement of the Heart in Pneumonia in Children. Am. Jour. Med. Sc., 1927, clxxiv, 448.

The author examined forty children with pneumonia in order to determine the position of the heart. In sixteen of the children he found a displacement of the heart to the affected side, sometimes very decided, more often of moderate degree. The cardiac displacement is a temporary one only. The lung is sometimes clear before the heart has resumed its normal position. If there is delayed resolution, the return of the heart is delayed as well. In at least two of the children, the heart returned to its place before the lung was entirely clear. The author believes that the cardiac displacement is dependent chiefly upon overdistention of the healthy lung. Small atelectatic areas in the affected lung may play some part in drawing the heart to the affected side.

Weiss, Soma, and Herrmann, L. Blumgart: Studies on the Velocity of Blood Flow. VIII. The Velocity of Blood Flow and Its Relation to Other Aspects of the Circulation in Patients With Pulmonary Emphysema. Jour. Clin. Investigation, 1927, iv, 555.

In order to estimate the relative significance of circulatory insufficiency and the disordered gaseous exchange of pulmonary emphysema producing a cough, dyspnea, and lowered vital capacity in patients with doubtful signs of cardiac pathology, the authors have studied the velocity of blood flow in twenty-five patients by means of measuring the arm to arm circulation time of an intravenous injection of "radium C." This method appeared particularly suited to the study of pulmonary emphysema because it neither postulates normal gaseous exchange nor requires cooperation on the part of the patients. The method measures not only the pulmonary circulation time but also simultaneously the velocity of blood flow from the elbow to the right auricle.

In twenty-one of the twenty-five patients studied the velocity of the blood flow was within normal limits. The normal, or even slightly increased, velocity of the blood flow, particularly in those patients who had many of the symptoms and signs of severe circulatory failure, such as conspicuous weakness, cyanosis and dyspnea, shows that pulmonary emphysema alone is sufficient for the production of these symptoms and signs. With two exceptions, the vital capacities of the patients studied were moderately or greatly reduced. The average venous pressure was 6.8 cm., which is slightly lower than 7.3 cm., the

average venous pressure found in sixty-five normal subjects. Four patients showed a velocity of blood flow slightly slower than normal. These patients with pulmonary emphysema were completely incapacitated in contrast to patients with cardiovascular disease, showing but slight restriction of muscular activity. The authors believe that in this group of patients in which the blood flow was slightly retarded and the venous pressure elevated, pulmonary emphysema was complicated by circulatory failure.

Letulle, Maurice, Labbe, Marcel, Heitz, Jean: Arterial Calcification in Diabetics. Arch. d. mal. du coeur, September, 1927, p. 577.

The authors reviewed the literature on radiographic visibility of peripheral arteries. In certain diseases, atheroma, senile gangrene, syphilis, and Paget's disease, slightly increased opacity may be found. In Bright's disease, in hypertension, and in thrombo-angiitis, this is rarely found. In diabetes more than in any other disease, radiographic evidence of arterial change is present. The x-ray findings precede the changes in pulse pressure, but are especially marked where the blood pressure changes are present and where intermittent claudication is a symptom. On the other hand, the prognosis, based on the x-ray findings, is less reliable than that of the sphygmomanometer, as a considerable amount of change in the arterial trunks is possible without any change in the circulation.

The authors report in detail the findings in a case of diabetic gangrene involving the feet, where the blood cholesterin was markedly elevated and the blood calcium slightly raised; x-rays showed calcification of the vessels of the thighs, legs, feet, and forearms. Subsequent autopsy examination showed changes in those same vessels and also in visceral arteries, e.g., splenic. The cerebral vessels were unaffected. The changes consisted in a thickening of all three coats of the vessel. Cholesterin was present in considerable amounts in some of the vessels, and calcification was present in varying amounts in the three coats, especially in the media, rarely to any extent in the intima. In the right radial artery, the calcium content was 64 times the normal amount.

The authors conclude that in diabetics the peripheral vessels are the most affected, thus differing from syphilitic arteritis, and that in spite of the extensive changes the lumen of the vessels usually remains patent. Although the deposition of cholesterin is accompanied by a cholesteremia, no such relation exists between the calcium deposit and the blood calcium which may be slightly raised or may be reduced. The deposition of the cholesterin and calcium and the thickening of the coats of the vessels occurs along parallel lines. Calcification of media may occur in three forms: (1) as fine particles lying among almost normal tissue; (2) as foci of atheroma with irregular deposition of

cholesterin and calcium, especially in the media not far from the internal limiting membrane; (3) as plaques of calcium without histological structure included in a strand of sclerosed tissue.

Lukin, N., and Frey, D.: Total Heart-Block in a Child Eight Years Old. Arch. Pediat., 1927, xliv, 647.

The authors report a case of complete heart-block in a girl of eight years, who was watched over a period of five months. The etiology was doubtful. The diagnosis of a "weak heart" had been made five years previously, by a physician, during an attack of influenza. No cardiac symptoms were present. A tonsillectomy and appendectomy had been uneventful. Physical examination showed a child of normal nutrition and appearance. The heart was slightly enlarged to the left. Pulse rate varied between 43 and 60 beats per minute, reaching the upper figure after exercise. Blood pressure was 72 systolic, 45 diastolic. Blood examination, urinalysis, and Wassermann were negative. The electrocardiogram showed a complete dissociation. The authors discuss the mechanism in heart-block.

Weber, Klement: Paroxysmal Arterial Hypertension During the Course of a Subacute Meningococcus Meningitis. Arch. d. mal. du coeur, September, 1927, p. 598.

The author reports a case of a woman, aged twenty-eight years who, during the course of a meningococcus meningitis had paroxysmal attacks of arterial hypertension. The diagnosis was confirmed at autopsy when an internal hydrocephalus was found with recent multiple foci of softening of the brain and cord, especially of the latter. No pathology of the circulatory system was demonstrated. During the crises described, the patient had severe headache and vertigo, bradycardia, and an incompressible pulse. The blood pressure rose from about 140 to 224 mm. of mercury, but fell to the previous figure within thirty minutes. The author considers the simplest interpretation of the mechanism of paroxysmal hypertension to be that of Laignel Lavastine, who states that it is due to a change in the vasomotor tone. Three factors may be considered as initiating this change: (1) Transitory increase in adrenalin in the blood. This is supported by the case of paraganglioma described by Labbe, Tinnel, and Daumer. (2) Irritation, peripheral or central, of the vasomotor apparatus. This is shown to occur in lead poisoning, where the solar plexus is irritated; in experimental observation of cerebral compression, and in the rise in tension which occurs on stimulation of the cervical cord after section of the medulla. (3) It may be reflex due to strong stimulation of sensory nerves. The author considers that his case fell into the second group and that the hypertension was due to irritation of the vasomotor

mechanism. No known explanation was given, however, why a constant anatomic lesion, such as was found, should provoke a *crisis* of hypertension.

Mahaim, Ivan: Contribution to the Study of Bundle-Branch Block. Ann. dé med., 1927, xxii, 213.

The author reported five cases of bundle-branch block which conformed to the findings in the experimental work on this condition. A review of the literature was given, the authors criticizing those reported cases which did not conform to experimental findings.

The first case, a woman fifty-four years old, had cardiac decompensation and a right bundle-branch block, depending on advanced pulmonary tuberculosis. Clinically, a presystolic gallop rhythm was present. The electrocardiogram showed a left ventricular predominance, the enlarged ventricular complexes notched in all three leads, and the T-wave diphasic. The picture was that of right bundle-branch block. Ouabain was given at first, with some improvement. Later venesection with the use of digitalis was employed with success. During digitalis therapy, extrasystoles from the right and left sides were visible. Between courses of digitalis, no extrasystoles occurred on the blocked side. This indicates that digitalis acts on the terminations of the bundle.

The second case was that of a woman of fifty-seven years with pronounced decompensation. The electrocardiographic study showed a rapid fibrillation rate of 180. Digitalis was without effect, and quinidine was tried. As a result, a reduplication of the first heart sound developed and the electrocardiogram showed a left preponderance with prolongation and notching of the ventricular complex. An increase in the quinidine caused the signs of block to disappear, but on a further increase the reduplicated sound and the electrocardiographic changes returned. On stopping the use of quinidine, the tachycardia returned, and the first sound did not show reduplication. Quinidine was, therefore, again given (4.6 gm.) with a return of the reduplicated first sound of the right bundle-branch block. A further dose of 1.4 gm. of the drug slowed the rate to 80, the reduplication disappeared, and the sinus rhythm was established with a normal jugular pulse. The quinidine was continued with a small and diminishing dose, with continuation of the sinus rhythm. The author emphasized that bundle-branch block was no contraindication to quinidine therapy. The coincidence of reduplication of the first sound and the electrocardiographic evidence of bundle-branch block suggested that the former was of diagnostic importance.

The third case, a man aged seventy-five, with syphilitic aortic stenosis, showed left preponderance with enlargement and notching of the ventricular complexes and diphasic T-waves. A clinical diagnosis of

syphilitic arteritis of the nutrient vessels to the right branch of the bundle was made. This proved to be true at autopsy.

The fourth patient was a girl of eighteen with a history of palpitation for from eight to ten months. The electrocardiographic tracings showed the presence of auricular extrasystoles. Quinidine was given in increasing doses with the result that the extrasystoles diminished in number, and in the extrasystolic cycles, the P-R interval was increased and the ventricular complex took the form of a levocardiogram. No symptoms accompanied this change. Bigger doses of quinidine caused entire disappearance of the ectopic beats. Here the quinidine depressed the conductivity in the right branch of the bundle which, therefore, could not transmit to the right ventricle the premature excitation wave coming from the auricle. The A-V node and the left branch were unaffected. The final increase in the dose of quinidine prevented altogether the occurrence of the auricular premature beat.

Case 5 was that of a man, aged twenty-seven years, with rheumatic mitral and aortic disease and decompensation. A diffuse block was present throughout the left branch of the bundle, as shown by right preponderance and an increase in the size and notchings of the QRS complex. Digitalis produced a marked bradycardia, with A-V block and auricular fibrillation. The ventricular complexes ceased to show the notching and became pure dextrocardiograms.

One assumed that the left bundle-branch block was here further exaggerated by the inability of the myocardium to transmit to the left ventricle the excitation wave coming from the right ventricle. The author concludes that bundle-branch block is not a contraindication to the use of digitalis or quinidine, although extrasystoles may appear in cases of bundle block by the use of the former, and bundle-branch block may result from the latter drug. The clinical evidence of complete bundle-branch block consists of a reduplication of the first heart sound; a presystolic gallop rhythm accompanying a reduplication is less common.